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A.M.A. ARCHIVES OF OTOLARYNGOLOGY

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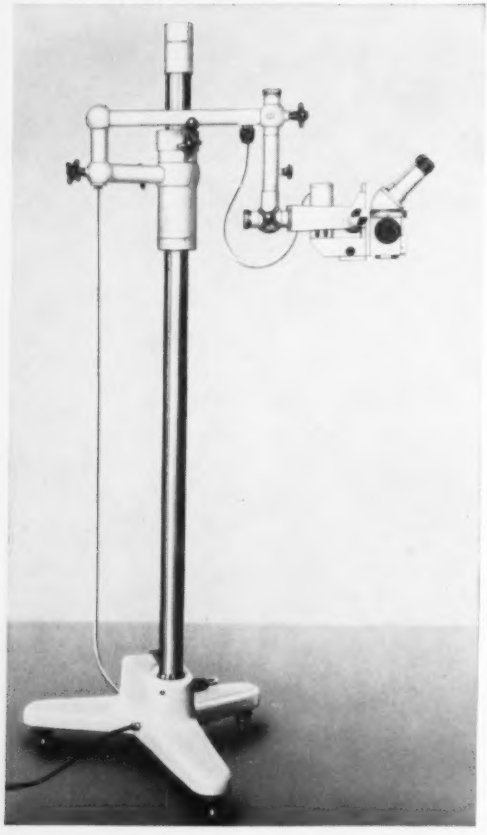

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Archives of Otolaryngology

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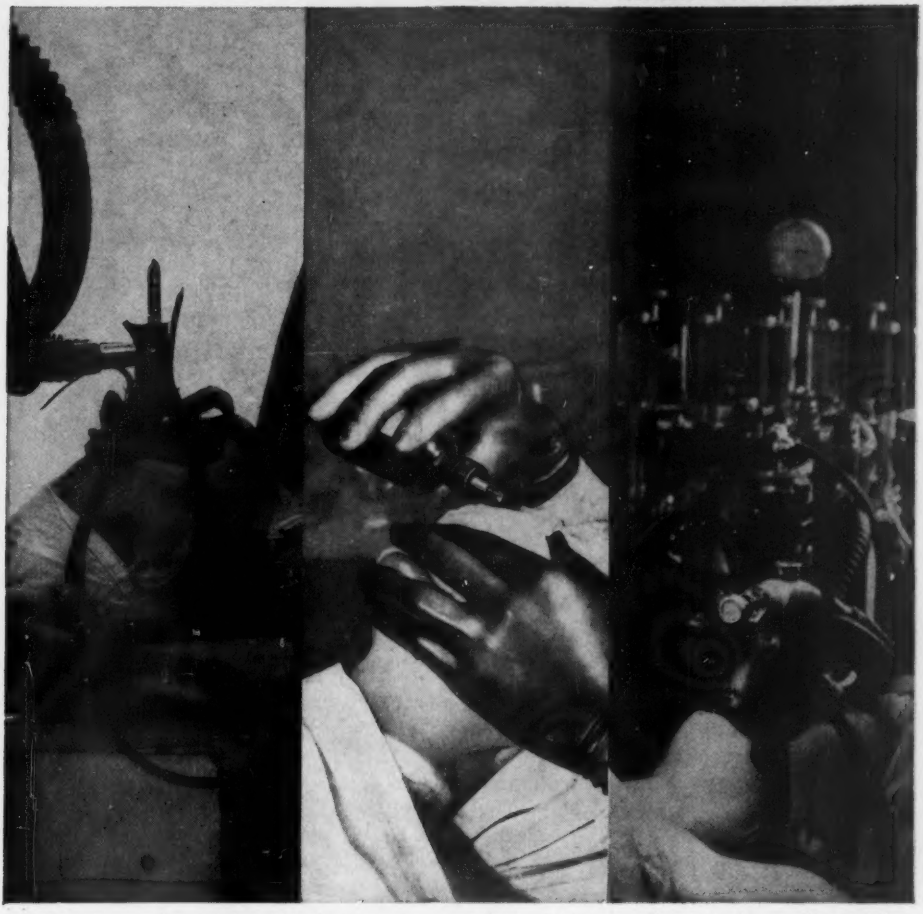

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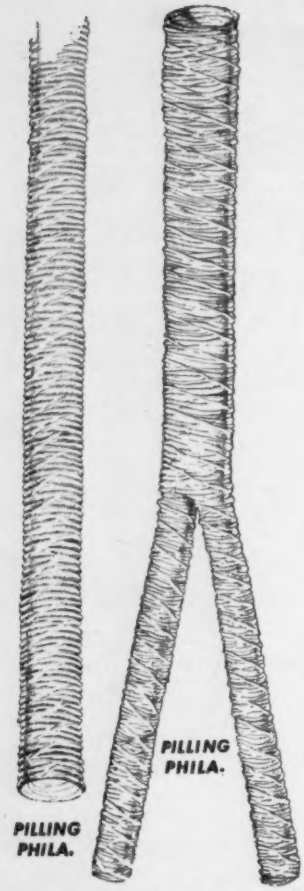

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1. DeBakey, M. E., Cooley, D. A., Morris, G. C. Jr., Collins, H.: Arteriovenous Fistula Involving the Abdominal Aorta, Annals of Surgery, 147:646 (May, 1958)

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Repair of Ossicular Defects with Ostamer

JOSEPH SATALOFF, M.D., Philadelphia

Ostamer * is a polyurethane foam which has been used successfully as a bonding agent in fractured and diseased bones.[1-3] In its presently available form, Ostamer is prepared by mixing together at surgery a prepolymer (prepared by reacting a polyol with an aromatic polyisocyanate) with a catalyst (a mixture of alaphatic tertiary amines and water) in the ratio of 4:1. The resultant mixture is a thick, extremely adhesive substance from which carbon dioxide and heat are liberated. On hardening, it swells to twice the original volume to form a very light porous plastic.

Ostamer produces little or no tissue reaction at the time of application, according to published reports on animals and humans. The oldest human implant was performed three years ago, and as yet no evidence of toxicity has been detected.

In view of the adhesive properties, light weight, and apparent absence of toxicity, the possibility of using Ostamer in the repair of defects in the ossicular chain was considered. The present preliminary report describes my experiences with this material in the middle ears of cadavers and of living humans.

Materials and Methods

Ostamer, which is at present available for experimental use only, was supplied in two sterile containers, one containing approximately 60 cc. of viscous prepolymer and the other approximately 15 cc. of viscous catalyst. When used in the repair of long bones most of the contents of the smaller container are poured into the prepolymer and the mixture is stirred rapidly for about 30 seconds with a sterile tongue blade. At this time, a white opacity is noted and the jar becomes very warm. The mixture is applied to the bone as quickly as possible and is allowed to set for 20 minutes, during which time any excess is removed with a scalpel.

In repairing ossicular defects, approximately 1 cc. of catalyst was added to 4 cc. of prepolymer and the two were stirred with a small probe. Only 15 seconds were then available in which to place one small drop of the material in the proper place before it became too hard to use.

Ostamer was used at first in the ears of 25 cadavers in which defects were created in various parts of the ossicular chain. After this "practice period," it was used in two living patients.

The first patient was a 14-year-old youth who had suffered from a marked hearing impairment for 9 years after a right simple mastoidectomy. On two successive occasions, during the two years prior to the present attempt, I had tried to repair a large defect between the head of the stapes and the absent long process of the incus which had probably been inadvertently damaged during the mastoidectomy or through a myringotomy. On neither occasion was there real success in repairing the defect with the use of small pieces of bone or fibrous tissue applied between the head of the stapes and the end of the incus. Prior to application of the Ostamer during the third surgical procedure, care was taken to maintain a dry operative field and to remove as much mucosa as possible from the bone ends. The mucosa was removed from the end of the incus with a curved curette placed on the undersurface and from the head of the stapes with a pick. A drop of Ostamer was placed on the stapedial head

Submitted for publication April 20, 1959.

From the Department of Otolaryngology, Jefferson Medical College.

* Ostamer is the trademark of the Wm. S. Merrell Co., Cincinnati, for its brand of polyurethane foam.

NAME _____

DATE	RIGHT EAR AIR CONDUCTION							LEFT EAR AIR CONDUCTION					
	250	500	1000	2000	4000	8000		250	500	1000	2000	4000	8000
1-6-59	40	50	60	25	40	40	PRE.						
1-24-59	5	20	50	30	45	45	POST.						

Figure 1

and the end of the incus pressed into the drop until it hardened.

The second patient was a 45-year-old woman who claimed unilateral insidious deafness for three years but denied having had ear infections. The eardrum was opaque and had a large posterosuperior marginal perforation. During one attempt to correct the conductive deafness, the entire long process of the incus was found to be missing and a large space existed between the remainder and the head of the stapes. The stapes was normally mobile in the oval window. In an attempt to approximate the incus and stapes, the entire incus was inadvertently dislocated and was removed. In replacement of the eardrum, an attempt was made to close the perforation by sliding the posterior canal wall into contact with the other side of the perforation and also to adhere this sliding graft to the head of the stapes. This was only partially successful, resulting in a moderate hearing improvement which regressed after several months. A small perforation also remained, through which it was possible to palpate a part of the stapedial head.

In reoperation on this ear to apply Ostamer, the eardrum was elevated and detached from the stapedial head. This resulted in a larger perforation in the eardrum, through which the stapes was visible. The posterior flap was consequently replaced and the head of the stapes debrided with a pick through the perforation. A large drop of Ostamer was placed on the stapedial head and, as far as possible, the edges of the perforation were incorporated into the sticky Ostamer as it hardened.

Results

In the cadavers, after defects had been created in various parts of the ossicular chain, repairs were made with Ostamer. In several ears, the entire incudi were removed and large gaps were left between the mallei and the stapes. Small pieces of hardened Ostamer (previously prepared) were shaped to approximate the defects. These were then glued in place by applying fresh Ostamer to the ends of the hardened Ostamer. There was no real difficulty in making it stick to the mallei and the heads of the stapes. With minimal ingenuity, it was possible to repair ossicular defects by using fresh Ostamer to glue pieces of hardened Ostamer to the exposed end of the remaining ossicles.

In the first patient, the gap between the head of the stapes and the incus was filled with Ostamer. Results of a preoperative audiogram and one taken one week postoperatively indicate a substantial hearing improvement (Fig. 1). There was little or no change at 2,000 cps or at higher frequencies.

In the second patient, the absence of the incus and a large perforation in the ear drum made columelization the procedure of choice. In preference to placement of a graft over this perforation, a drop of Ostamer was placed over the head of the stapes and the edges of the perforation were embedded into the Ostamer filling the defect. The improvement in hearing one week postoperatively is shown in Figure 2.

NAME _____

DATE	RIGHT EAR AIR CONDUCTION							LEFT EAR AIR CONDUCTION					
	250	500	1000	2000	4000	8000		250	500	1000	2000	4000	8000
11-11-58	50	60	50	60	60	50	PRE.						
2-13-59	15	25	25	55	55	45	POST.						

Figure 2

Comment

It is fruitless to speculate on the amount of hearing improvement that might have occurred after using Ostamer in ears of cadavers. It is noteworthy, however, that in every case the continuity of the ossicular chain was restored.

In the first case, it was a simple procedure to place a drop of Ostamer on the head of the stapes and push the incus into the drop until it hardened. Despite much previous practice, however, the drop of Ostamer was excessive and overlapped the incus, as seen in the model in Figure 3. This may account for the lack of hearing improvement at the high frequencies.

In the second case, some difficulty was encountered in getting the Ostamer to remain soft until it stuck to the stapes and the edges of the perforation were embedded. Here, too, an excessive amount was used due to miscalculating the expected expansion after the Ostamer hardened. A major difficulty in filling so large a perforation with Ostamer is the rough surface left exposed to the external canal. There is a tendency for debris to stick to this surface both at the time of surgery and afterward.

Since pressure on the Ostamer tended to move the stapes, extreme caution was exercised in pushing the Ostamer exposed through the ear drum. This Ostamer was actually a columella direct to the stapes.

Fig. 3.—Ostamer (bone glue) connecting the incus and stapes.

In its present form, Ostamer has a number of disadvantages in the repair of ossicular defects. There is insufficient time available for applying the mixture to the ossicles before it hardens. The need to adjust the microscope, to select the proper-sized drop, and to place it effectively sometimes takes so much time that the Ostamer frequently hardens and a new batch has to be mixed. It would be preferable for the Ostamer to take several minutes to harden or, even better, to remain slightly elastic so that it could act like a joint in certain situations and also be able to be trimmed when necessary. In its present bulky preparation it is sheer waste to use Ostamer for otological work. It should be available in small amounts already autoclaved.

Prior to applying the Ostamer, it is essential to have a dry field free of blood and to remove as much mucosa as possible from the bone ends to be adhered. The latter can be accomplished with a curved curette placed on the undersurface of the incus and with a pick on the head of the stapes. In application of the Ostamer, care is needed to avoid applying it anywhere except to the intended site. Considerable practice is advisable in cadavers before using it in human ears. This is particularly necessary because it is extremely difficult to trim any excess or to remove Ostamer inadvertently stuck to nearby parts of the ear. When Ostamer is being applied to a stapes, it must be remembered that the Ostamer cannot be removed once it hardens.

I have also used Ostamer on the proximal end of polyethylene tubing when it is used to replace the stapes in mobilization procedures. By using a small amount of Ostamer, it is possible to assure firm attachment of the tube to the incus. The tube should be made slightly shorter to accommodate the expanding Ostamer if the tube is placed under the incus.

Summary

Ostamer was used successfully in restoring the continuity of the ossicular chain after defects had been created in the ears of cadavers.

In two living patients, Ostamer was used with good results after conventional methods of restoring continuity of the ossicular chain had failed. In both instances, there was a substantial improvement in hearing.

While Ostamer in its present form has been used to advantage in the repair of ossicular defects, it would be desirable that the material be modified, if possible, to prolong the hardening time and increase the elasticity.

Dr. R. H. McMaster, of The William S. Merrell Company, and Dr. H. Menduke aided in this investigation.

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The Indiscriminate Use of Masking in Bone-Conduction Audiometry

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Many authorities contend that the opposite ear should always be masked during bone-conduction audiometry in order to eliminate the possibility that the bone-conducted sound, crossing the skull, will be heard in the nontest ear.

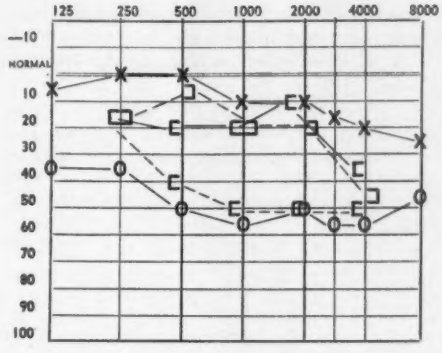
Other investigators, while cognizant of the omnipresent danger of cross hearing to the nontest ear, nevertheless feel that masking should be used only if the patient actually refers the test tone to the nontest ear, that as long as he hears the tone on the proper ear, it is neither necessary nor desirable to mask the opposite ear.

Still other investigators are somewhat apprehensive over the largely unexplored problem of how a rather loud noise in one ear may affect threshold responses in the other ear in many clinical patients, irrespective of the cross hearing problem. They remind us that just as it is not possible to stimulate only one cochlea by bone conduction so also it is not possible to mask out only one cochlea with air-conducted noise. The noise inevitably activates not only the desired cochlea but the entire complex of higher auditory pathways as well. The extent to which it may even affect the opposite cochlea via the newly discovered efferent pathways is a matter for future research.

In any event, the following case illustrates and highlights some of the as yet unanswered problems in bone-conduction audiometry by showing how the use of masking on the nontest ear yielded a completely erroneous bone audiogram.

Report of a Case

The Figure shows the audiogram of a 46-year-old white woman with a moderate unilateral loss



Audiogram of a 46-year-old white woman with unilateral otosclerosis. Broken line denotes bone curve on right ear with masking noise on left ear. Solid line denotes bone curve on right ear without masking noise on left ear.

first noted about six months previously. Vertigo, nausea, tinnitus, and fluctuation in acuity were all denied.

The otologic examination was essentially negative. Both drums were normal and intact. Tuning fork tests performed independently by four separate otologists all agreed in suggesting a sensorineural, or perceptive, loss on the right ear. The Rinne was negative at 250 but positive at 500, 1,000, and 2,000 cycles. The Schwabach was normal at 250 cycles but shortened at 500, 1,000, and 2,000 cycles. The Weber was lateralized to the right ear at 250 but to the left ear at 500 and 1,000 cycles.

Two separate bone curves for the right ear are shown on the audiogram. The lower curve, represented by dashed lines, was obtained while the left ear was masked with complex noise (Beltone 15-A) at a level sufficient to reduce sensitivity on the left ear by approximately 50 db. It is commonly considered in clinical practice that this would be the minimum amount of masking necessary to ensure that bone-conducted sounds applied to the right mastoid would not be heard on the left ear.

The bone curve, joined by solid lines, was obtained without masking of noise on the left ear. All of these responses were referred by the patient to the right, or test, ear. At no time were bone stimuli delivered to the right mastoid reported as

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heard on the left ear. Still, this unmasked bone curve has all the ear marks of a "shadow" response from the left ear, particularly in view of the shift in bone acuity produced by contralateral masking.

In spite of this compelling set of data, all other auditory tests on the right ear pointed toward a conductive loss. SISI scores were low; the Békésy audiogram was negative; the alternate binaural loudness balance test showed no recruitment, and speech discrimination was excellent, with a PB Max of 94%.

Accordingly, surgical exploration was undertaken. The right middle ear was entered with the usual stapes mobilization approach. The overhanging bone at the level of the annulus was very extensive. With use of the Ziess operating microscope, a considerable amount of the bony rim was removed, to give excellent exposure of the footplate area. There was no question, after observation and palpation of the incudostapedial joint and the footplate directly, that the stapes was completely fixed by otosclerosis. The otosclerotic bone appeared to be at a mature stage of growth, i. e., white, with little vascularity, and had involved the entire footplate, except for a small central area. The round window niche was open, and the entire mucosa and remaining structures appeared healthy and in normal relationship.

Air-conduction audiometry with the flap in place and rotated anteriorly showed no appreciable change from the preoperative audiometry. Extensive palpation and percussion at the footplate level failed to obtain mobility. The ear was closed without an improvement in hearing.

There was no apparent explanation derived from the surgical procedure to explain the preoperative discrepancies obtained by audiometry and tuning fork.

Comment

This case illustrates one of the serious pitfalls inherent in the indiscriminate use of contralateral masking during bone-conduction audiometry. Too little is known of the extent to which the mere presence of noise on one ear may yield a completely false picture of bone acuity on the other ear.

While results such as these emphasize the need for further research on the problem of a completely satisfactory method for bone-conduction audiometry, the clinician will continue to face situations in which the use of a contralateral masking noise is indicated.

Our purpose in this report is simply to stress that he must do so with extreme caution and with full awareness of the limitations inherent in this technique.

Hearing Clinic, Northwestern University.

Pathology of Ossicles in Chronic Otitis Media

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In the course of surgical procedures performed in patients with chronic otitis media, the ossicles of the middle ear, particularly the incus, are found often to be markedly involved in the disease process. An investigation of the pathological changes found in these bones was carried out in the anticipation that such a study would aid in the determination of the mechanism of this destructive process and might help in the decision as to the type of surgical procedure most likely to give the best result.

Some of the ossicles removed at surgery during the past three years at the University of Illinois, Illinois Eye and Ear Infirmary, were collected and included in this study. These specimens were prepared, after proper decalcification and dehydration, for serial section and embedded in either celloidin or paraffin.

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Normal Structure of Ossicles

Normal specimens of the malleus and incus for comparison were obtained by preparing sections of those ossicles removed in the course of fenestration surgery. In these specimens the periosteum is found to consist of a narrow fibrous layer containing numerous capillaries. The mucosal covering of the head of the malleus and the incus is usually a simple squamous epithelium. It is a very thin sheet, closely adherent to the periosteum (Fig. 1). Occasionally the epithelium is higher, to give a cuboidal appearance (Fig. 2). Eggston and Wolff [1] describe this epithelium as made up of simple nonciliated cells having no basement membrane; Ash and Raum [2] call them flattened cuboidal cells, and Maximow and Bloom [3] state that they are simple squamous epithelium.

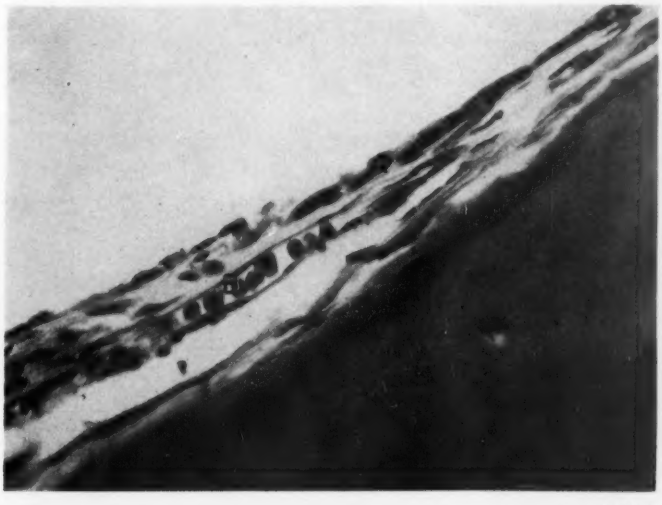

Fig. 1.—Periosteum of normal ossicle covered by epithelium and containing thin-walled capillary. *A*, simple squamous layer. *B*, capillary lined by endothelium.

Fig. 2.—Periosteum of normal ossicle covered by a cuboidal epithelium. *A*, cuboidal epithelium. *B*, bone.

The ossicle itself is made up of firm compact bone, layered in Haversian systems, which are irregular in shape owing to the contour of the ossicles.[1] The bone contains many marrow spaces. These are found in the head of the malleus and the body of the incus. According to Eggston and Wolff the stapes contains no marrow. The osseous spaces are more numerous in the incus than in the head of the malleus. The spaces themselves contain a network of fine connective tissue which supports a network of capillaries, made up of a single layer of endothelium (Figs. 3 and 4).

The bone structure of the malleus and the incus is, in many respects, very comparable with the long bones elsewhere in the body, according to Lempert and Wolff.[4] As

Fig. 3.—Head of normal malleus showing compact bone spaces. *A*, compact bone of Haversian system. *B*, large marrow space.

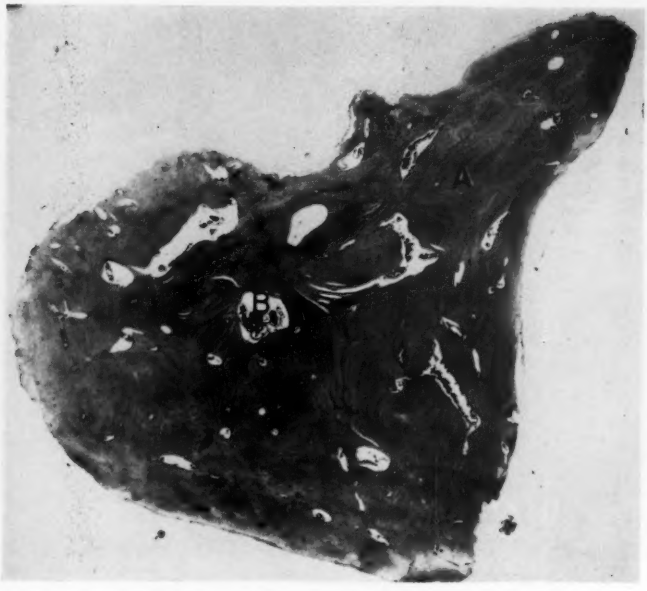

Fig. 4.—Normal incus showing bone and bone spaces. *A*, compact bone. *B*, osseous space containing blood vessels.

in other bones, there are Haversian systems, Haversian canals, lacunas, canaliculi, and centrally located marrow spaces. The malleus and incus are miniature long bones which are more or less isolated in the middle ear cavity. They are completely enveloped by mucosa that is continuous with the mucosal lining of the middle ear.

Histopathological Changes

Histological examination of the removed ossicles demonstrated certain fairly uniform changes. There was often a metaplastic alteration of the epithelium to that of a stratified squamous type (Fig. 5). Occasionally the epithelium was not seen. This

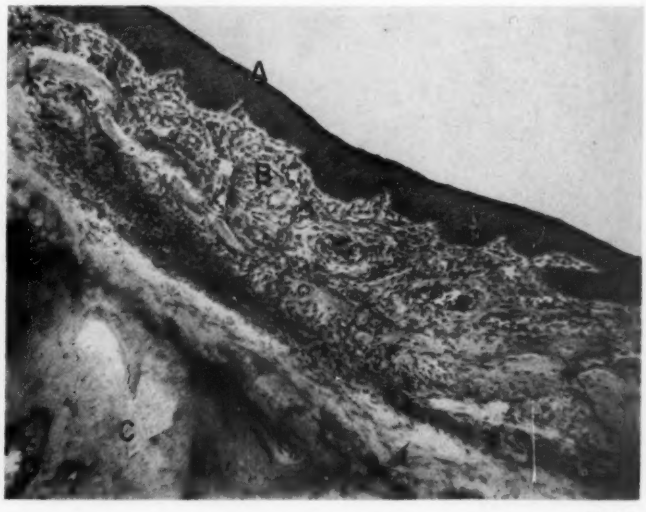

Fig. 5.—Metaplasia of the epithelium to a stratified squamous type. *A*, stratified squamous epithelium. *B*, thickened periosteum. *C*, bone of ossicle which appears to be dead. *D*, thickened vessel wall.

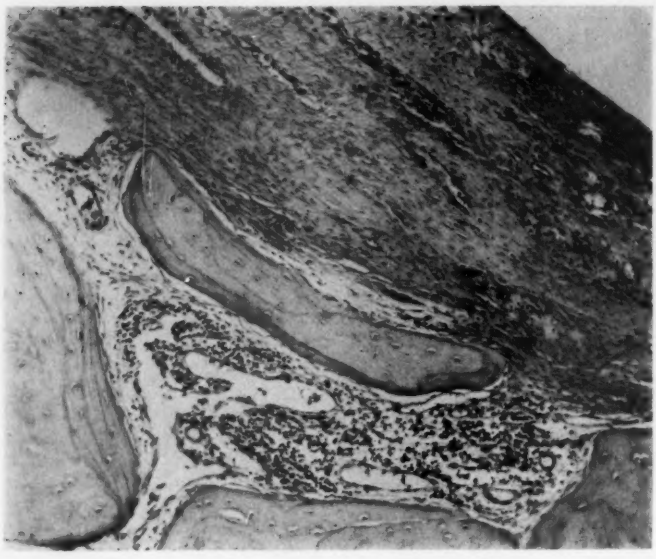

Fig. 6. — Periosteum, markedly thickened, consisting of dense connective tissue.

is thought to have been due to alterations in the processing and to preparation of the specimens. The metaplasia is a protective mechanism resulting from the chronic irritation of the inflammatory process. The ossicles, particularly the incus, were frequently found to be embedded in granulation tissue. The periosteum was markedly thickened and made up of dense connective tissue (Fig. 6). Granulation tissue, composed of fibrous tissue, newly formed blood vessels, and round-cell infiltrates, were observed external to the bone.

The most characteristic finding observed was the change in the osseous spaces themselves. These alterations were particularly prevalent in the incus and most frequently in the long process, near the region of its bony destruction. The reticular network bearing capillary vessels was considerably altered. The spaces contained clusters of round cells, small lymphocytes, and plasma

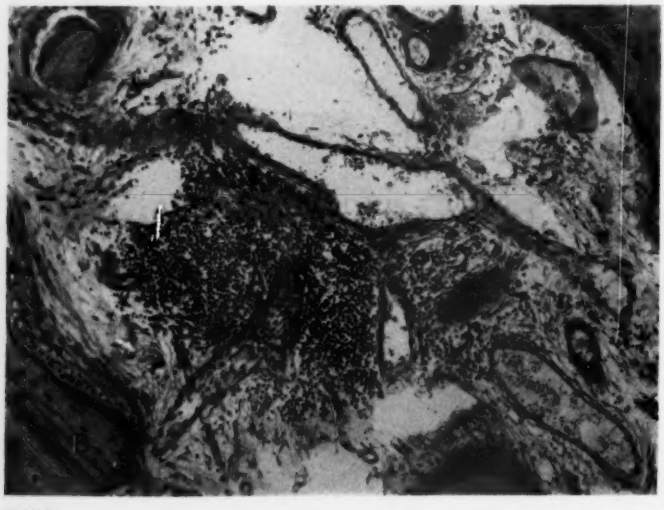

Fig. 7.—Marked round-cell infiltration, in bone space, surrounding a thickened blood vessel. *A*, cluster of round cells. *B*, lamellated bone.

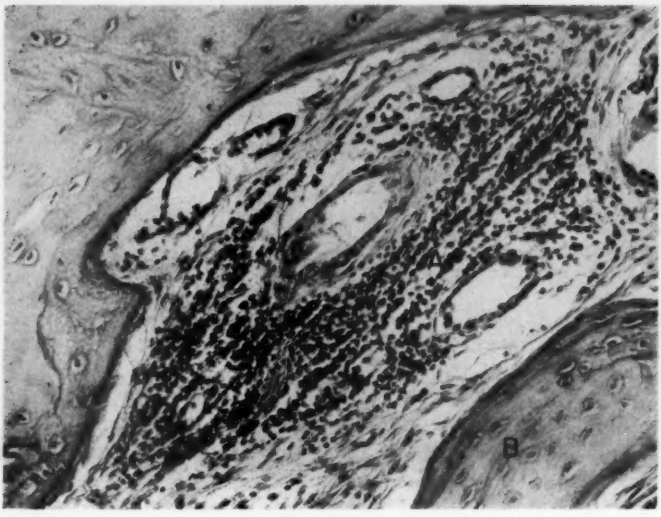

Fig. 8.—Cluster of round cells in bone space. *A*, round cells surrounding thickened blood vessel. *B*, osteocyte.

cells. There were occasional polymorphonuclear leukocytes found. The round cells were often seen to be clustered around the blood vessels (Figs. 7 and 8). In other specimens the round cells completely filled the spaces (Figs. 9 and 10). Fibrous tissue often occupied the spaces in the bone. This tissue was found in areas where there were masses of round cells in some instances, while in other portions the fibrous tissue was seen to be filling the entire space (Figs. 11 and 12).

Blood vessel changes were prominent in some of the ossicles studied. There appeared to be an increase in the number of capillaries. The endothelial lining of the vessels was sometimes found to be thickened (Fig. 13).

The bone itself was involved in the areas surrounding the marrow spaces in some instances but appeared normal in other ossicles. Osteocytes were often absent from their lacunae, and the normal architecture had disappeared. In some instances a spicule

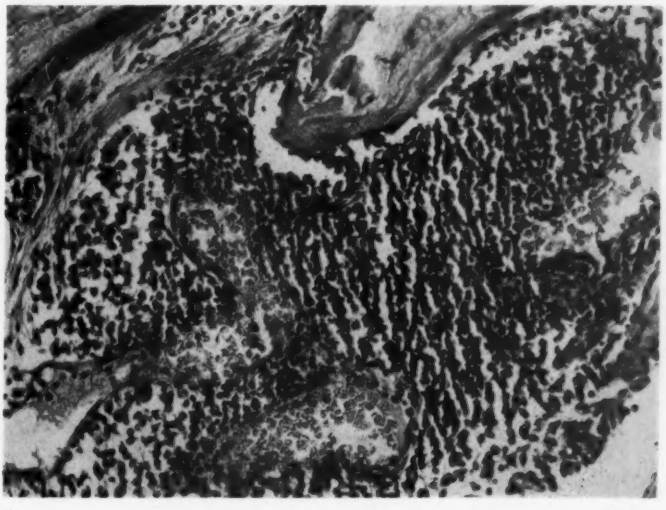

Fig. 9.—Mass of round cells almost completely filling bone space in the incus.

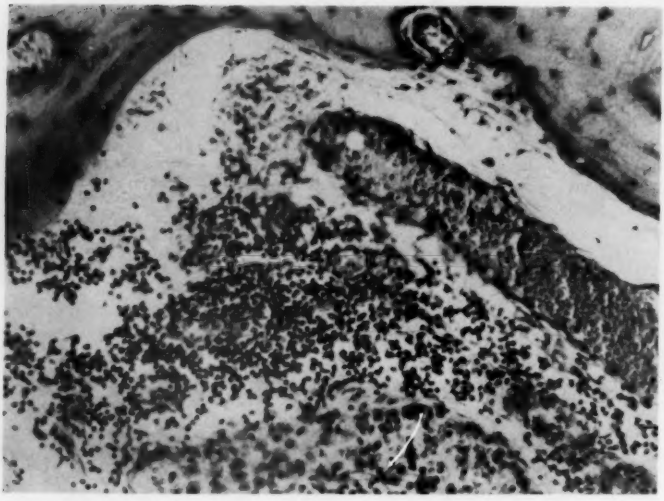

Fig. 10.—Mass of round cells in bone space.

of sequestrated bone was seen (Fig. 14). Osteoclasts were seen in some areas of the bone spaces (Fig. 15). In other areas osteoblasts were noted (Fig. 16). Destruction of large area of bone in the ossicle was occasionally encountered (Fig. 17).

The loss of some portion of the incus, particularly of the long process, frequently seemed to have been produced by inflammation of the surrounding tissue. In one instance the cholesteatoma was pressing on the long process, thereby causing its destruction (Fig. 18). In other areas granulation tissue was found to be surrounding the eroded tip of the incus, while the adjacent bone spaces were filled with fibrous tissue and round cells. In such instances external inflammation as well as alterations in the bone surrounding the spaces apparently contribute to the destructive process (Figs. 19 and 20).

Comment

It has long been recognized that the ossicles of the middle ear may become diseased

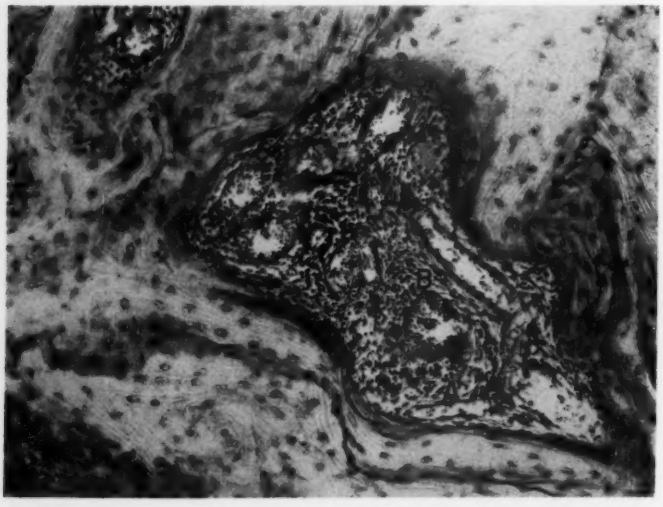

Fig. 11.—Fibrous tissue and round-cell infiltration seen in bone space. *A*, cluster of round cells. *B*, fibrous tissue in bone space.

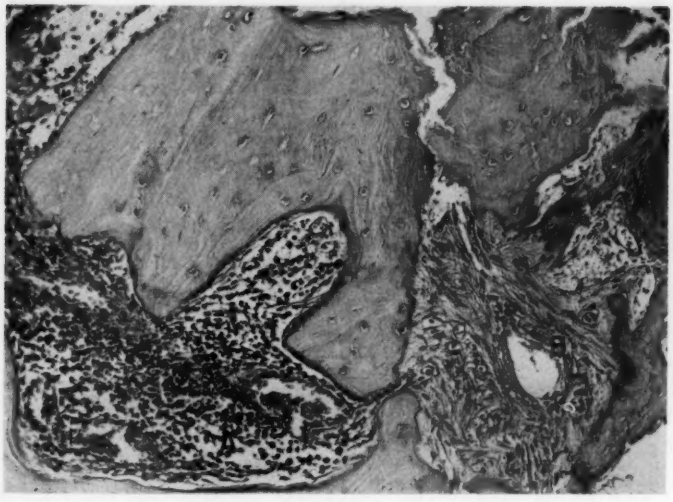

Fig. 12.—Round cells filling space on the left, while fibrous tissue almost completely occupies spaces on the right. *A*, bone space filled with round cells. *B*, fibrous tissue occluding osseous space.

in chronic otitis media. Schwartze[5] described caries of the ossicles, especially in attic involvement. With long-standing attic disease, the head of the malleus and the body of the incus will be involved. He attributes continuation of suppuration to this involvement.

Dingley and Zwiefach[6] found that in the presence of attic cholesteatoma the incus was more frequently eroded, while in other forms of chronic otitis media the malleus was more frequently involved, although the incus escaped or suffered only minor damage. The gross findings of the ossicles were recorded in a majority of patients at the time of surgery. Twenty of these had cholesteatoma in the mastoid process, epitympanum, or mesotympanum. Grossly, there was destruction of a portion of the incus in 18 cases and destruction of some part of the stapes in 9 instances. In 11 patients there was no cholesteatoma present, but granulation tissue in the mastoid process, epitympanum, or mesotympanum was found

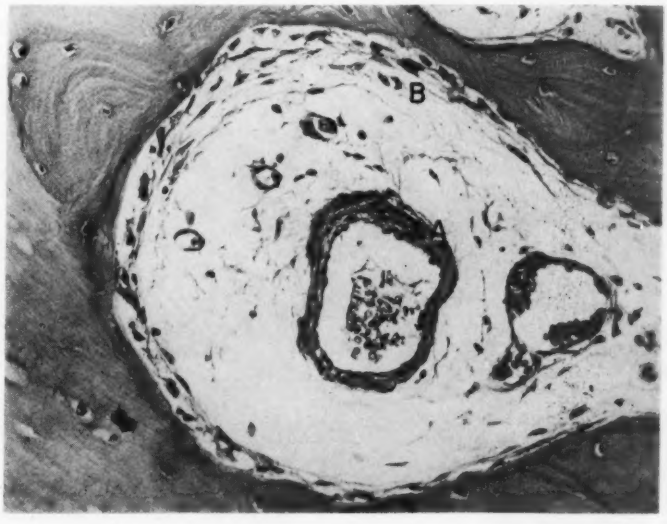

Fig. 13.—Thickened wall of blood vessel in bone space. Osteoclasts are seen near the bony wall. *A*, vessel with a thickened wall. *B*, osteoclasts.

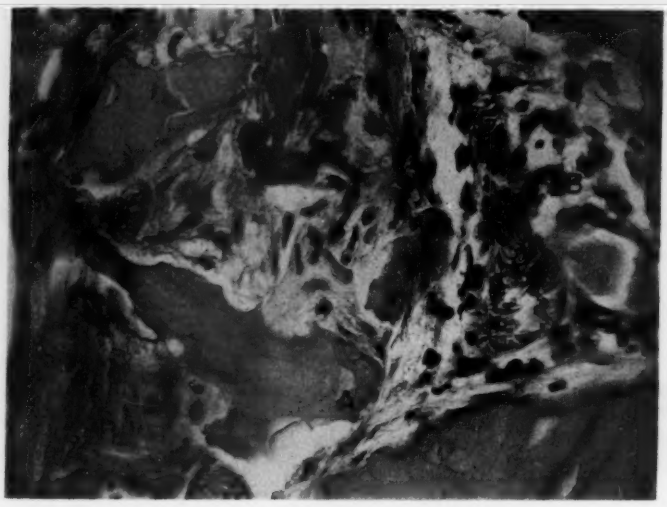

Fig. 14.—Spicules of sequestrated bone seen in edge of bone space, containing fibrous tissue and round cells. *A*, bone sequestra showing loss of normal structure. *B*, lymphocytes. *C*, osteoclast.

at surgery. A part of the incus was eroded in eight specimens, and a portion of the stapes destroyed in two cases. If would appear from the present study that the ossicles may be involved with or without the presence of a cholesteatoma.

The gross changes in the ossicles in chronic otitis media seemed essentially to be found in the long crus and the lenticular process of the incus and in the capitulum and crura of the stapes. The normal appearance of the malleus and incus are shown (Fig. 21). The long process of the incus was destroyed at the very tip in some cases, while in others the entire process had disappeared (Figs. 22-25).

When the incus was involved in the process, the incudostapedial joint was destroyed. In involvement of the stapes, the crurae were sometimes partially eroded and occasionally they were completely destroyed. In one instance the footplate was also destroyed.

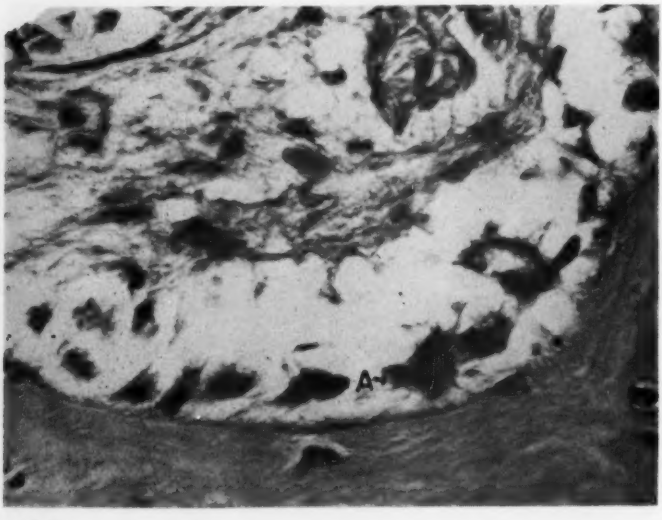

Fig. 15.—Osteoclasts lining bone in space. *A*, osteoclast.

Fig. 16.—Osteoblasts in bone space. *A*, osteoblast.

Histopathological studies of the ossicles of the middle ear in chronic otitis media have not often been carried out. There appears to be little of this type of study in the literature, especially in association with clinical data. Grippaudo [7] recently presented a study of 55 ossicles removed in 41 cases of chronic suppurative otitis media. The changes that he described parallel those reported in the present study, concerned with 31 patients with chronic otitis media treated surgically in which the ossicles were removed and examined.

In each of the 31 cases that have been examined, there were pathological changes found in the ossicles, particularly in the incus. The bone spaces, in each instance, evidence alterations from the normal. These include fibrous tissue in the spaces; infiltration by clusters of round cells; increase in the vascular bed; thickening of the blood vessel walls; destruction of bone adjacent to

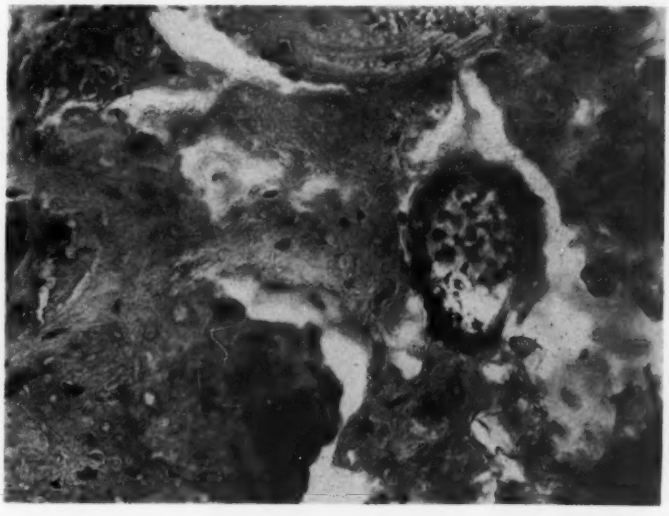

Fig. 17.—Large area of necrosis of bone seen in incus. There is a thickened vessel in the center of this destruction. *A*, thickened wall of blood vessel. *B*, bone showing loss of normal structure.

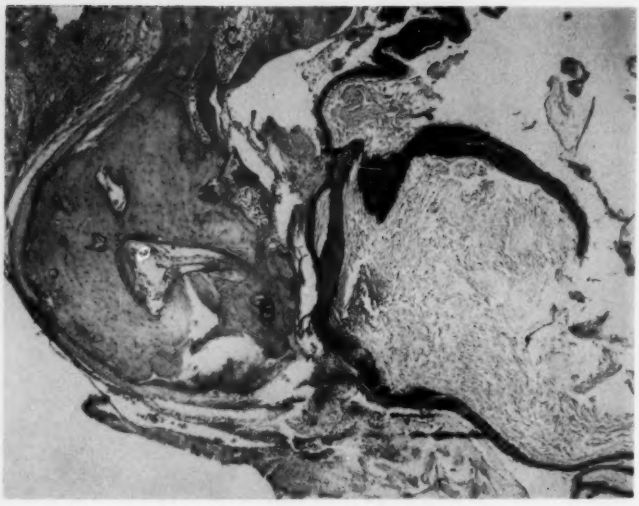

Fig. 18. — Cholesteatoma pressing upon the long process of the incus. Round cells can be seen in the bone spaces. *A*, stratified epithelium forming wall of cholesteatoma. *B*, erosion of bone of long process of incus. *C*, marrow space filled with round cells.

the marrow spaces, and an attempt at repair of the bone by osteoblastic activity. Changes observed in the bone and marrow spaces correspond to the changes that are found in chronic osteomyelitic involvement in other bones. Therefore, in the cases studied, it can be stated that a chronic osteomyelitic process has developed in the ossicles of the middle ear, particularly in the long process of the incus.

Tumarkin [8] noted that the incus hangs suspended in midair and that its anatomical connections are tenuous in the extreme. He feels that its blood supply at best must be precarious and in the presence of cholesteatosis is liable to become inadequate. Nager and Nager,[9] by following individual blood vessels in serial sections of temporal bone, observed that a single branch of the anterior tympanic artery, which they have named the ossicular artery, supplies the outer ossicles. This ossicular artery divides into two branches, one supplying the malleus and the other the incus. The vessel to the long process of the incus has convolutions as it traverses the marrow spaces. The mucosal

Fig. 19.—Long process of incus being destroyed by external inflammation and changes in the bone space. *A*, granulation tissue surrounding eroded bone. *B*, bone space filled with fibrous tissue. *C*, osteoclast.

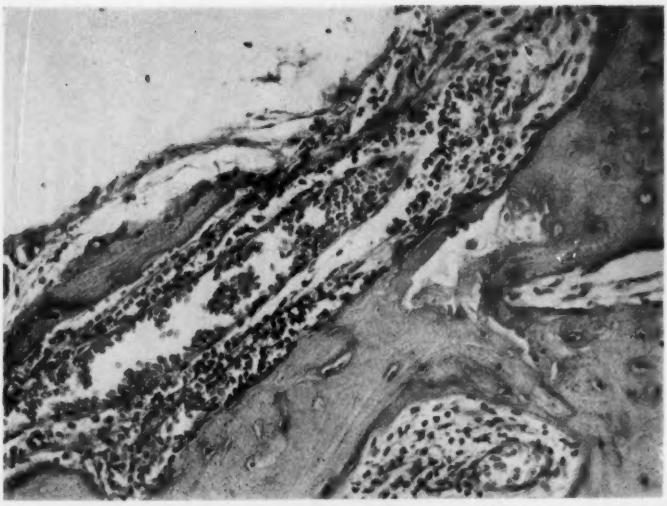

Fig. 20.—Necrosis of bone due to inflammation of the periosteum and in the bone spaces.

Fig. 21.—Photograph of the normal appearance of the malleus and incus.

Fig. 22.—Erosion of entire long process of incus. Normal malleus.

Fig. 23.—Erosion of large portion of long process of incus. Normal malleus. *A*, edge of eroded area of long process.

Fig. 24.—Erosion of long process of incus. Normal malleus. *A*, edge of eroded portion of long process.

Findings and Pathology of Thirty-One Cases Examined

No.	Age	History	Drum Findings	Audio	Surgical Findings	Ossicles	Pathology
1	10	Otorrhea many yr.	Anterior, attic, & large cent. perforation	40-45-50-45-45-45	Large cholesteatoma in epitympanum & middle ear	Long process of incus absent	Stratified squamous epithelium; marked infiltration of bone spaces; dead bone ajacent to some spaces
2	45	Mastoidectomy 32 yr. previously	Drum absent cholesteatoma in attic	40-45-50-55-85	Cholesteatoma in antrum, epitympanum, & middle ear	Marked erosion of incus; footplate only remaining of stapes	Fibrous tissue filling bone spaces; edges of spaces eroded
3	39	Discharge from both ears since childhood	Attic & small posterior marginal perforations	35-35-40-45-35-35	Cholesteatoma in epitympanum	Synostosis of head of malleus and body of incus; stapes eroded	Fibrous tissue filling many of bone spaces
4	17	Discharge right ear for 5 yr.	Granulations filling middle ear; attic filled with cholesteatoma	30-45-45-30-30-55	Cholesteatoma in attic & over facial canal	Malleus & incus intact	Stratified squamous epithelium; many lymphocytes & plasma cells in bone spaces; some dead bone ajacent to spaces
5	28	Bilateral discharge for 14 yr.	Central perforation; attic perforation	35-35-30-20-35-0	Cholesteatoma in epitympanum & mesotympanum	Incudostapedial joint destroyed	Fibrous tissue & ound cells filling many bone spaces
6	13	Hearing loss; no history of otorrhea	Superior marginal perforation	40-55-50-35-45-40	Antrum filled with granulations; small cholesteatoma in mesotympanum	Erosion of long process of incus	Stratified squamous epithelium; round cells filling bone spaces
7 & 8	5	Discharging ears for 2 yr.; has cerebral palsy	Large central perforations with granulations	Unable to do one	Left: small cholesteatoma in epitympanum	Long process of incus eroded	Fibrous tissue & round cells fill spaces; bone eroded from outside & in bone spaces
					Right: large cholesteatoma filling entire mesotympanum	Long process of incus gone; only footplate of stapes remained	Stratified squamous epithelium; fibrous tissue & round cells fill bone spaces; osteoclasts seen; cholesteatoma eroding long process
9	26	Discharge from right ear for 3 mo.	Anterior attic perforation with cholesteatoma	20-25-35-30-40-20	Granulation tissue in antrum & epitympanum	Long process of incus eroded	Round cells completely filling bone spaces
10	15	Discharging ears for many yr.	Large perforation inferior with granulations covering attic	70-65-70-70-70-65	Granulations in antrum & attic; cholesteatoma in attic	Not recorded	Fibrous tissue & round cells fill bone spaces; blood vessel walls thickened
11	46	Aural polyps removed repeatedly	Canal filled with large polyp	50-75-70-50-50-70	Sclerotic bone; granulation tissue throughout	Long process of incus eroded	Fibrous tissue filling bone spaces; blood vessels in bone spaces thrombotic; round cells & fibrous tissue in spaces near erosion of malleus

12	26	Ear infection for 8 yr.	Canal filled with large polyp	65-60-80-75-75-100	Antrum & middle ear filled with granulations	Long process of incus destroyed	Bone spaces filled with round cells & new blood vessels
13	65	Otorrhea for 25 yr.	Attic destruction with posterior perforation	60-70-80-90-95-100	Granulations around ossicular chain; cholesteatoma in mesotympanum	Long process of incus destroyed	Some round-cell infiltration in bone spaces; granulations around end of long process
14	35	Bilateral discharge for 4-5 yr.	Posterior superior perforation	Not found	Large cholesteatoma from epitympanum to mastoid	Long process of incus almost completely destroyed	Bone spaces toward long process of incus filled with round cells; bone ajoining some spaces destroyed; osteoclasts seen
15	8	Discharge for many yr.	Attic perforation with granulations & cholesteatomatous material	55-45-30-30-30-40	Large cholesteatoma in antrum, epitympanum, mesotympanum, & hypotympanum	Long process of incus absent; stapes completely eroded	Increase of fibrous tissue & round cells in bone spaces
16	30	Discharge for 23 yr.	Small posterior attic perforation	25-15-35-40-40-40	Large pocket of cholesteatoma matrix in attic	Long process of incus destroyed; crura of stapes eroded	Marked round-cell infiltration of bone spaces
17	22	Constant foul discharge for 2 yr.	Large attic perforation filled with white material	20-35-50-15-30-20	Large cholesteatoma filling antrum & entire middle ear	Long process of incus destroyed	Some round-cell infiltration of bone spaces
18	12	Discharging ear for 2 mo.	Large round polyp from anterior superior attic perforation	Not found	Granulations throughout	Normal	Aseptic necrosis of areas of bone; bone spaces filled with some round cells; erosion of bone by granulation tissue outside of ossicles
19	9	Discharging ears for a long time	Foul discharge and granulations filling canal	Not found	Granulation tissue filling whole middle ear		Marked round-cell infiltration of bone spaces; osteoclasts seen in spaces; erosion from outside & inside
20	16	Discharging ears for many yr.	Large central perforation & attic destruction	35-40-60-40-40-60		Normal	Stratified squamous epithelium; some granulations in bone spaces
21	23	Pain and discharge from ear for 2 mo.	Large polyp filling canal	40-45-40-45-45-10	Granulations in attic, antrum, & mesotympanum	Long process of incus absent; only footplate of stapes remains	Round cells filling most bone spaces throughout; perivascular infiltrations

Findings and Pathology of Thirty-One Cases Examined (Continued)

No.	Age	History	Drum Findings	Audio	Surgical Findings	Ossicles	Pathology
22	28	Discharging ear for 13 yr.	Attic perforation with thick pus	30-25-30-35-25-45	Small cholesteatoma in mesotympanum	Incus eroded	Stratified squamous epithelium; destruction by pressure of granulations on outside of bone; marked round-cell infiltration of bone spaces with destruction of bone about them
23	44	Discharging ear for a long time	Destruction of pars tensa with posterior or attic wall destruction	55-50-40-30-40-40	Scleratic mastoid filled with granulations	Long process of incus destroyed	Stratified squamous epithelium; granulation tissue filling bone spaces, with aseptic necrosis about some spaces
24	34	Attacks of vertigo; no history of discharge	Crusty area in attic region	35-45-45-35-55-20	Some granulations in attic; no cholesteatoma	Long process of absent incus; head & crura of stapes absent	Some perivascular round-cell infiltration in bone spaces; osteoclasts seen in spaces; some aseptic necrosis of bone
25	10	Discharging ear all her life	Posterior marginal perforation large aural polyp	25-30-40-20-40	Granulation in middle ear; cholesteatoma in epitympanum	Long process of incus destroyed	Thickened blood vessel walls, fibrosis, & round cells in bone spaces near long process of incus; some aseptic necrosis
26	14	Discharging ears since childhood	Large posterior-inferior perforation with polypoid granulation tissue	35-40-45-40-45-55	Cholesteatoma matrix filling mastoid & epitympanum	Long process of incus, crura of stapes, & portion of malleus eroded	Stratified squamous epithelium; fibrous tissue & round cells in bone space
27	39	Discharging ear for 9 yr.	Polyp filling canal, attic perforation	30-25-30-30-15	Granulation tissue filling epitympanum & mesotympanum	Long process of incus eroded	Marked fibrosis & round cells in bone spaces; erosion of bone in spaces & from outside
28	50	Intermittant discharge for many yr.; hearing loss	Tympanic membrane plastered to promentory	40-65-65-60-70-↓	Cholesteatoma in mastoid; granulations in mesotympanum	Long process of incus eroded	Infiltration of round cells in bone spaces in long process of incus
29	27	Discharge for 1 ½ yr.	Entire drum destroyed	50-45-50-50-60-30	Granulations in attic; cholesteatoma mesotympanum & hypotympanum	Destruction of long process of incus; destruction of head & crura of stapes	Round cells & fibrous tissue in bone spaces near long process of incus with some dead bone in this process
30	12	Discharge for 6 yr.	Large central perforation with purulent secretion	5-35-35-35-40	Cholesteatoma in mesotympanum	Incus partially destroyed; malleus eroded; only footplate of stapes remains	Some round-cell infiltration of bone spaces near long process of incus; long process eroded, probably from outside pressure
31	62	Discharging for many yr.	Many small polyps & granulations filling entire canal	35-35-40-45-70-70	Granulation tissue in middle ear	Long process of incus partially destroyed	Round-cell infiltration in bone space; long process; minimal changes

Fig. 25.—Erosion of portion of long process. Some irregularity of malleus. *A*, edge of eroded process of long process of incus. *B*, eroded part of malleus.

network over the long process of the incus seldom sends vessels into the bone. Because of this meager vascular supply described by the Nagers, chronic inflammation of the marrow spaces through which the blood vessel courses may lessen the vascularity of the long process and be a factor in its subsequent destruction and dissolution.

Interference with the integrity of the ossicular chain in chronic otitis media, with or without the presence of cholesteatoma, must be a factor in the selection of the surgical management of this disease. The hearing loss does not necessarily correspond to the destruction of the long process of the incus and the head and crurae of the stapes (Table). Zollner [10] believes that a cholesteatoma may form a connection between damaged ossicles, so that if it is removed there must follow a loss of hearing. It would seem, therefore, that the incudostapedial area should be carefully examined in every surgical case, in order to determine the type of repair that is best to be carried out.

Inasmuch as a chronic osteomyelitic process was found in the area of the incudostapedial joint in each of the cases that were examined, it would seem inadvisable to allow this bony area to remain in the interests of correct surgical care of these patients. Ormerod and McLay [11] emphasized that the first consideration of treatment should be eradication of the disease and surgery should be radical enough to achieve this object. If destruction of the long process of the incus is seen under microscopic examination at the time of surgery, a reparative procedure should be performed which should include the removal of the osteomyelitic bone.

Summary

The ossicles in 31 patients with chronic otitis media have been examined histologically. The architecture of the normal malleus and incus is similar to that found in other long bones of the body. The changes observed in the ossicles examined correspond to the findings in a chronic osteomyelitic process. If a chronic osteomyelitis is present in these ossicles, the surgical correction of this disease must include the removal of these diseased ossicles.

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Bell's Palsy

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The term "Bell's palsy" is commonly used to indicate the idiopathic type of facial paralysis. Of late, there seems to be general agreement that this disorder is the result of vasospasm of the nutrient vessels of the nerve with the usual effect of anoxia, that is, increased permeability of the vessel wall with consequent accumulation of interstitial fluid, thus, edema. Because the facial nerve is enclosed in a rigid bony canal the so-called tunnel syndrome is produced with its vicious circle of vasospasm, anoxia, edema, and pressure on vessels with further anoxia.

Audibert, Mattei, and Paganelli [1] appear to have been the first to present this hypothesis. They considered that the only reasonable explanation of Bell's palsy is dysfunction of the autonomic nervous system, which produces spasm, thrombosis, or embolism in the vessels supplying blood to the facial nerve. They felt that the facial nerve is particularly susceptible to vasomotor disturbances because its blood supply comes in part from the middle meningeal artery and in part from the stylomastoid artery. Müller [2,3] pointed out that vasomotor disturbances are particularly likely to occur at the junction of arteries. Audibert and co-workers stated that the exciting stimulus bringing on the vasospasm was chilling of the face and ear on the same side.

Kettel [4] accepted the hypothesis of Audibert and co-workers and supported it with the findings at operation on 50 patients who had Bell's palsy. In all his patients he found granular decay and necrosis of bone without cellular infiltration. His operative findings were confirmed by Cawthorne [5] and by Sullivan and Smith.[6] He concluded that the primary and central feature of Bell's palsy is a disregulation of the circulation, which probably takes place near the stylomastoid foramen. The result is ischemic paralysis and ischemic bony necrosis. He felt that in patients with Bell's palsy there was a "cold allergy" related to the autonomic disintegration, which Petersen [7] pointed out to be present in Ménière's disease.

The hypothesis has received experimental support in the work of Coassolo.[8] He studied the electrical excitability of the facial nerve after refrigeration in two groups of rabbits. The first group had previously been sensitized. The second group consisted of nonsensitized rabbits. In the second (nonsensitized) group of rabbits the clinical findings were completely normal and the electric excitability of the facial nerve was completely unchanged. In the first (previously sensitized) group of rabbits evidence was obtained of static and dynamic insufficiency of the refrigerated nerve. In the first group of rabbits this study also demonstrated early involvement of the nerve bundles (half a day after refrigeration) before evidence of insufficiency and progressive recovery of the nerves with disappearance of the evidence of the alteration in about 30 days.

The Facial Nerve

As stated before, the relation of the facial nerve to the Fallopian canal is of importance in the development of the syndrome of Bell's palsy, and its structure and relations are of importance in the differential diagnosis of lesions of the seventh cranial nerve.

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Tamari [9] described the seventh, or facial, nerve as a mixed nerve consisting of a large motor and a smaller sensory portion.

The motor fibers rise in the pyramidal cells in the cerebral cortex and run to the nucleus of the seventh cranial nerve in the pons by two tracts, one to the opposite side and the other to the same side, the latter of which is the larger. From the nucleus of the seventh nerve in the pons the motor fibers, after partly circling the abducent nucleus, appear between the spinal tract of the fifth cranial nerve and the nucleus of the sixth cranial nerve. Tschiassny [10] pointed out that the motor fibers for the orbicularis oris muscle rise in the hypoglossal nucleus and pass to the facial nerve. Thus in supranuclear and nuclear lesions of the facial nerve the movement of the orbicularis muscle is preserved. In lesions of the upper motor neurons the action of the frontalis, the orbicularis oculi pars palpebrarum, and the corrugator supercilii is preserved because these three muscles have bilateral cortical representation.

Motion of the entire face from emotional stimuli is also preserved in the presence of lesions of upper motor neurons and cortex because of tracts outside the pyramidal tract which connect the nucleus in the pons with hypothetic emotional centers in the thalamus, corpus striatum, and substantia nigra. Tschiassny [10] pointed out that in lesions of the upper motor neurons ipsilateral hemiplegia is usually present and paralysis of the third nerve is not infrequently present.

After passing between the spinal tract of the fifth and the sixth cranial nuclei, the facial nerve emerges at the upper end of the medulla oblongata in the depression between the olivary and restiform bodies.

The sensory portion of the facial nerve rises from the geniculate ganglion situated on the knee-shaped bend of the facial nerve as it lies in the facial canal. The central processes of its ganglionic cells pass inward with the motor portion of the facial nerve as far as the internal auditory meatus and then separate as the pars intermedius or nerve of Wrisberg. At the bottom of the meatus the facial nerve enters the facial canal (aqueduct of Fallopius) and follows the windings of that canal through the temporal bone to the lower surface of the skull. The nervus intermedius lies between the motor portion of the seventh cranial nerve and the acoustic nerve. The separate bony canal for the facial nerve begins at the fundus of the inner acoustic canal going through the porus acusticus internus above the crista transversaria. The facial canal then departs from the axis of the inner acoustic canal at an angle of from 30 to 50 degrees, sloping cephalad and ventrad for a distance of about 3 mm., to enter the elliptic groove for the geniculate ganglion, which forms the sensory nucleus for the facial nerve. The nerve then runs caudad and dorsally to the supratympanic portion, which starts cephalad to the oval window and lies in a semicanal attached to the caudal border of the horizontal semicircular canal and terminates above the exit of the stapedial muscle. The next adjacent segment of the facial canal, which is called the "post-tympanic portion," is directed at first slightly posterior and downward and then changes to a downward direction below the middle of the horizontal semicircular canal. The vertical portion then begins about 2 mm. below the bulge of the horizontal canal and runs downward in a slightly lateral slanting direction for 10 to 13 mm. The exit of the canal, the stylomastoid foramen, is infundibuliform, owing to the division of the facial nerve into its main branches. It lies just ventral to the crest in the tip of the mastoid formed by the bulging inward of the digastric fossa.

In the course of the facial nerve through the Fallopian aqueduct, it is joined by sensory fibers of the great superficial petrosal nerve, which rise directly from the geniculate ganglion and carry sensory impulses from the posterior part of the nose and the posterior part of the palate. Other sensory

fibers are closely associated with the motor portion of the facial nerve almost to the stylomastoid foramen, where they leave the canal by the iter chordae posterius, which opens on the posterior wall of the tympanum close to the attachment of the drumhead. They then arch across the tympanic cavity, lateral to the long process of the incus and medial to the manubrium mallei, to leave the cavity by the iter chordae anterius at the inner end of the Glaserian fissure. The chorda tympani then unites with the lingual nerve to supply taste to the anterior two-thirds of the tongue. Costen, Clare, and Bishop [11] have recently shown that the chorda tympani also carries common sensation.

Haynes [12] found that variations in the course of the facial nerve are rare except for the junction of the chorda tympani with the facial nerve, which is so variable that in Haynes' opinion its localizing value when involved in lesions of the facial nerve is small.

Tschiassny,[13] however, expressed the opinion that the anatomic peculiarities of the facial nerve and Fallopian canal are of extreme value in differential diagnosis of the location of lesions of the nerve, and he described syndromes of facial paralysis. These are essentially the supranuclear (upper motor neuron) lesions, lesions in the course of the facial canal, and lesions peripheral to the stylomastoid foramen. The five levels in the course of the facial nerve which he identified were the infrachordal, the infrastapedial, the suprastapedial, the transgeniculate and the suprageniculate. He suggested appropriate tests for lacrimation, phonophobia, and dysgeusia to confirm such localization.

Sullivan and Smith [6] stressed the importance of the blood supply to the facial nerve. They stated that the large or small nutrient vessels on reaching the nerve divide into ascending and descending branches which course in the epineurium. The vessels concerned are arranged predominantly in a longitudinal fashion in the perineural or intrafascicular connective tissue, making the arteries supplying a nerve into a continuous longitudinal system. Since the recovery of a degenerated nerve depends on adequate nutrition, preservation of an adequate vascular supply is of great importance in surgery on the seventh cranial nerve. They warned against carelessly stripping the nerve from its bed, as this may interfere with adequate nutrition.

Clinical Considerations

Although the anatomy of the facial nerve is of importance in differential diagnosis and "topagnosis," these are not the most pressing questions which a physician confronted with a given case of Bell's palsy must necessarily ask himself. To most of us physicians these are as follows: 1. Is the lesion severe enough to produce complete degeneration of the neuraxons in the vicinity of, and peripheral to, the lesion? 2. If degeneration of the nerve is complete, what are the chances of complete recovery? 3. What treatment gives the best chance of restoration of function with the minimum of deformity?

Wechsler [14] stated that mild facial paralysis with minimal or no electrical changes lasts two to four weeks.

Ford and Woodhall [15] observed that in patients with Bell's palsy with complete degeneration of the facial nerve there is never a return to completely normal power to move individual muscles of the face on the affected side. When the eye is closed, the corners of the mouth move and vice versa. These are called "associated movements" or "synkinesis" and are attributed either to branching of axons in regeneration or to fiber interaction in a pathologically altered region of a nerve.

Williams, Lambert, and Woltman [16] pointed out that in addition to these associated movements contracture of the muscles on the affected side might take place. Spiller [17] attributed this to overstimulation of affected muscles so that complete relaxation is not obtained and the muscles kept in a contracted state gradually shorten.

A third common occurrence is the appearance of hemifacial spasm on the affected side. Features of hemifacial spasm comprise tonoclonic contractions of muscles from frontalis to platysma, notably those at the eye and the mouth. Irregularity in site, rhythm, and degree is the salient feature.

Ford and Woodhall [15] pointed out that these sequelae of Bell's palsy might occur, whether there had been any anatomic interruption in the continuity of the nerve or not, as long as degeneration of the fibrils had taken place.

Sullivan and Smith [6] stated that incomplete recovery occurred in 25% of their cases, and in reviewing 500 cases of facial paralysis Park and Watkins [18] arrived at a similar figure. It appears that 10% to 15% of patients who have had Bell's palsy have a persisting deformity which is cosmetically and psychologically of major importance to the patient.

It seems, therefore, that, if some method of screening could be found to separate patients without complete degeneration from those with it, the possibility of coming to a decision as to what line of treatment to adopt would be furthered greatly.

Unfortunately, however, it is impossible to foretell when degeneration of the neuraxon will take place. Failure of the nerve to respond to faradic stimulation takes place two to four days after the initial insult, the rapidity of the disappearance of the reaction depending on whether the injurious lesion develops over a short time or tends to increase more gradually. Strength-frequency curves may show something in as short a time as eight days, but muscle fiber will only begin to show action potentials of denervation fibrillation in an average time of two weeks. This two weeks is the critical period.

As Joly stated,[19] Bell's palsy clears up in the majority of cases in 10 days to 2 weeks, and this fact leads to the success of treatment that is instituted early, such as radiation therapy, heat, sodium salicylate, and intravenous infusions of histamine and procaine. If signs of beginning recovery are not evident at the end of two weeks, it is reasonable to assume that degeneration has taken place, and it can usually be demonstrated both by galvanic reaction of denervation (R. D.) and by electromyography with the appearance of denervation fibrillation potentials.

At this time, however, the changes in the nerve are irreversible except for the growing in of axons from above, and some sequelae are inevitable, as pointed out by Tickle [20] and by Duel.[21] For this reason, Sullivan and Smith [6] pleaded that Bell's palsy be regarded from the point of view of those who do not recover spontaneously, since in 25% of cases the present usual line of treatment results in complete failure and permanent disfigurement.

In discussing this paper, Lempert [22] stated that he would like to know whether more would be gained than lost by early operation, because he felt that if these patients are operated on damage to the nerve might occur. He stated, "Accidents occur to the best of us and the incidence of accidents in this surgery may outweigh the percentage that may not get the result if left alone." This dilemma faces every physician who has a patient with Bell's palsy and will not be resolved readily. Will neurolysis of the facial nerve before the end of the first week be for the greatest good of the greatest number, or should neurolysis wait until degeneration becomes evident? I have found that voluntary movements of the involved muscles will begin a few hours after early decompression, but the question remains whether recovery would have taken place had I delayed operation for another week. I have concluded that delayed operation with its usually minimal sequelae is the best course to pursue with the best interest of the patient in mind, as I have found that serious disfigurement will not occur in more than 2% to 3% of patients with Bell's palsy if this course is followed. The question then

arises as to when to operate on the small percentage of patients in whom recovery does not become evident.

Ballance,[23] Duel,[21] Tickle,[20] and others have depended on the faradic reaction as an indication that the neuraxons are degenerated. With this, however, Kettel,[4] Sullivan and Smith,[6] and others disagreed on the grounds that, although the nerve may be under sufficient pressure to prevent the passage of stimuli, recovery without degeneration may still be possible.

Kettel [4] advised decompression in cases in which signs of beginning mobility have not appeared after an observation for two months, because he found no patient, who obtained satisfactory spontaneous recovery in whom signs of returning mobility could not be found within this period. He also pointed out that some other undiagnosed condition, such as a neurinoma, might be present. He also advised decompression if the spontaneous recovery of mobility had ceased before complete restitution had been obtained and in persons who have had repeated attacks of Bell's palsy.

However, in electromyography we have a still more certain method of prognosis than is furnished by Kettel's empiric rule. Skolnik [24] stated that when the faradic galvanic tests show either full or partial reaction of degeneration further examination by electromyography is necessary. When with electromyography only action potentials of denervation fibrillation are found and all three areas of the face are sampled, these findings are consistent with the diagnosis of a complete neuraxis cylinder degeneration and a poor prognosis is evident. A mixture of fibrillation potentials (at rest) and motor-unit potentials (with effort) indicates a partial lesion and a more hopeful prognosis. Skolnik [24] found that the earliest signs of regeneration are the disappearance of fibrillation potentials and the presence of polyphasic or nascent units during effort.

A reasonable rule, therefore, is that if no sign of beginning regeneration is indicated by electromyography after two months, the facial nerve should be decompressed.

Treatment of Bell's Palsy

Collier [25] stated that muscles deprived of their nerve supply become flaccid and undergo progressive atrophy. Wasting is most rapid in the early stages after injury and is more rapid in young subjects. As the muscle fibers shrink, fibrous tissue proliferates. If reinnervation occurs before the fibrosis is complete, the atrophic changes are reversible, but the greater the interstitial fibrosis the less the ultimate recovery. Muscle atrophy is accelerated by chronic overstretching, which is brought about by the pull of the muscles of the sound side and by gravity.

Stretching of the muscles attached to the upper lip can be controlled by an intraoral splint. Folkins and MacLeod [26] described a simple intraoral prosthetic appliance consisting of a hook fixed to the upper molars and inserted into the corner of the mouth. It has the advantages of simplicity of construction, lack of discomfort, cleanliness, and effectiveness with a pleasant appearance.

Joly [19] stated that electrical treatment of Bell's palsy should be extremely delicate and that it is well to be aware that too intense or badly directed electrotherapy will end in increased contracture, "because for an injured nerve all causes of irritation constitute factors leading to contracture."

Gutmann and Guttmann [27] and Jackson and Seddon [28] presented evidence that galvanism begun as early as possible after onset retards atrophy and helps to maintain contractibility.

Collier [25] stated that fears that galvanism is responsible for contractures are unfounded. In her opinion the exact nature of contractures is obscure and they are an accompaniment of regeneration and are as likely to be prominent when no physical therapy is employed.

Hilger and Goltz [29] suggested that Bell's palsy is a medical emergency and that in-

travenous infusions of procaine give evidence of improved function. However, evidences of improved function are minimal or doubtful and can only be obtained, if at all, during the infusion. There is no evidence that such treatment diminishes the incidence of sequelae or speeds recovery.

Intravenous infusions of histamine have also been advocated for Bell's palsy. Evidence is lacking that such treatment accomplishes anything more than assurance to the patient that everything possible is being done.

Rothendler [30] reported seven cases of Bell's palsy treated with cortisone. In the one in which evidence of degeneration of nerve fibers was found, cortisone therapy produced no response. The other six patients (with no evidence of degeneration) were reported to have responded promptly to cortisone. Robison and Moss [31] reported on the effect of cortisone on two patients with Bell's palsy on whom tests for nerve fiber degeneration were not done. The dosage ranged from 150 mg. per day for the first 48 hours to 75 mg. per day for the last 10 days of treatment. They secured complete recovery in 2 weeks in the first case and in 17 days in the second. The cases reported are too few for conclusions as to the effect of treatment to be drawn, and inasmuch as no evidence of degeneration was present in any of them, one is inclined to be skeptical as to the value of this treatment in hastening recovery, although again it supports the morale of the patient and in such a short time can do but little harm.

Exercise.—Collier [25] stated that exercise should be prohibited until there is clinical evidence of active movements. The severest wasting that she encountered was in patients who had disregarded this elementary principle. She found that overaction of the sound side in efforts to move the denervated muscles increased the stretching. In her opinion the time for exercise has arrived only when voluntary movements have become visible. Exercises should be carried out with the sound side firmly controlled by the hand so that the weak recovering muscles can act without having to work against too strong a pull.

Surgical Treatment.—Although recovery from facial paralysis following a degenerative lesion of the nerve is always imperfect, Kettel [4] has demonstrated that, when evidence of recovery cannot be obtained or when recovery after beginning has stopped short of completion, decompression of the facial nerve in the Fallopian canal will produce maximal function with minimal sequelae.

I am inclined to agree with Maxwell [32] that decompression of the facial nerve is most readily done through a postauricular incision. After exenteration of the mastoid cells, the Fallopian canal is exposed by removal of the bone of the posterior wall of the external auditory canal from the fossa incudis to the mastoid tip. The mastoid tip may then be removed with exposure of the pes anserinus, and the lateral and posterior walls of the Fallopian canal may be removed with curet and bur. In my own cases the lesion has not been confined to the region of the stylomastoid foramen, but swelling, reddening, and ecchymosis of the sheath of the nerve have usually reached to the post-tympanic portion of the nerve and in one case involved the supratympanic portion. In this instance, the nerve was uncovered nearly to the geniculate ganglion by tunneling through the posterior bony wall of the external auditory canal. In none of my cases has evidence of necrosis of the bone of the mastoid or Fallopian canal been impressive, although in some of them cloudiness of the cell walls of the involved mastoid has been reported by the roentgenologist.

In one patient on whom decompression was done before evidence of nerve degeneration was obtained, returning motion in the face was evident on the morning of the first postoperative day and complete normal function was obtained by the end of the first week. In all the other patients oper-

ated on because recovery was not evident after two months, some asymmetry of the face due to wasting of the muscles and to associated or mass movements (synkinesis) was evident.

Summary

In a case of Bell's palsy, the otologist is faced with a dilemma. Because recovery without sequelae is never complete when axon degeneration has taken place, should the nerve be decompressed in the predegeneration stage to prevent sequelae? Such action would make every case of Bell's palsy a surgical emergency and subject every patient with "idiopathic" facial palsy to operation immediately after onset. Since otologists are not immune to occasional technical lapses, this would endanger the great majority of nerves that would spontaneously recover. It seems, therefore, that the greatest good to the greatest number of patients with Bell's palsy will result from delaying operation and decompressing the facial nerve only of patients who do not recover spontaneously.

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Vocal Rehabilitation of Paralytic Dysphonia

V. Vocal Symptomatology After Bilateral Loss of Abduction

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Dyspnea and Dysphonia

It is common knowledge that the loss of phonic *adduction* of the vocal cords disturbs the voice and causes *dysphonia,* whereas the loss of respiratory *abduction* interferes with breathing and produces *dyspnea.* Such truisms serve as a general explanation but do not account for all fine details of disturbed function.

When attempting a more complete definition of the various disabilities caused by laryngeal paralysis, we should remember that adductor paralysis is characterized primarily by variable degrees of dysphonia and by concomitant complaints of dyspnea. As has been shown in the preceding paper,[5] there exists an *inverted relationship* between the degrees of dysphonia and dyspnea: Dysphonia increases and dyspnea diminishes in direct proportion to the width of the glottis. Conversely, the loss of abductor function creates a severe degree of dyspnea, while the simultaneous vocal disability is of a secondary and less disturbing nature.

The same observations are made in all cases of *bilateral immobilization* of the vocal cords in median or paramedian position. This condition is also known as bilateral median stenosis, bilateral midline fixation, or bilateral abductor or posticus paralysis. It should be noted that we prefer the term of median immobilization because it describes fully what laryngoscopy reveals: The cords cannot move away from the position of phonation.

No information on the *cause of immobilization* can be obtained by mere inspection of the larynx. As is well known, the cords may be immobilized either by (1) a *paralytic* condition affecting the laryngeal nerves or (2) an *arthritic* process which affects the cricoarytenoid joints. The differential diagnosis must be made by palpation of the arytenoids during local anesthesia: If they can be moved by a probing forceps, their immobilization is due to nervous paralysis; however, if they cannot be moved passively, their fixation is due to arthritic ankylosis. Tschiassny [25] recently emphasized this distinction.

The problem of mechanical fixation was discussed also by Grotting and Pemberton,[13] who described the laryngeal disorders caused by acromegaly. Owing to the abnormal growth of the laryngeal cartilages, the vocal cords may become fixed in the midline. Additional changes in the mucous membranes, such as edema and hypertrophy, cause the vocal cords to become thick and plump, which accounts for the vocal changes. Consequently, the hormonal disorder of acromegaly produces *dysphonia* with a low, rough, coarse, and hoarse voice due to the altered shape of the cords, and *dyspnea* with inspiratory stridor as a result of their median fixation. Réthi [21] believes that both paralysis and ankylosis lead to median immobilization, because contracture and shrinkage of the adductors forces the cords into this position.

Interpretation of Bilateral Median Immobilization

In spite of the recent advances made in the understanding of the pathology of laryngeal paralysis, the condition of bilateral median immobilization still poses several

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questions. My previous papers [2-5] attempted to show the concensus of present opinions. It appears that the majority of authors favor the concept that *median position* of a paralyzed cord indicates *complete paralysis* of the recurrent nerve.

Pressman and Kelemen [20] stated that a bilateral median position represents an essentially complete bilateral paralysis of the recurrent nerves. These authors discussed the *sympathetic innervation* of the larynx. This nerve supply is derived from the superior and middle cervical ganglion and from the carotid plexus, via the perivascular nerve fibers of the superior and inferior thyroid arteries. There can be no doubt that this sympathetic supply is important for the maintainance of muscular tonus.

The influence of sympathetic innervation was investigated further by Hartenau and Schwetz,[15] who experimented with cats. Section of the recurrent nerve produced promptly the intermediate position of the affected cord. A few hours later the cord shifted to the median position. However, after a few weeks, the paralyzed cord became permanently immobilized in the intermediate position. The results were very different when section of the recurrent nerve was combined with the interruption of the superior and inferior laryngeal arteries of both sides. In these cases, the affected cord remained permanently in the midline position. Histologic analysis demonstrated severe degeneration of all intrinsic laryngeal muscles of both sides. This finding was obviously related to the bilateral interruption of the blood supply and of the perivascular sympathetic innervation. According to these animal experiments, it appears that the permanent position of paralyzed cords depends in part on the vascular supply and on the perivascular sympathetic innervation. This observation is important for those cases of bilateral laryngeal paralysis which develop as a result of thyroid or other cervical surgery.

In his monograph on laryngeal movements, Berendes [6] analyzed numerous recent contributions to this subject. He believes that complete laryngeal paralysis is followed by median position of the paralyzed cord. If tonic contracture of all intrinsic muscles develops after bilateral recurrent paralysis, both vocal cords will be immobilized in median position. However, he retains a guarded standpoint with regard to certain cases of *unilateral damage* to one recurrent nerve which is followed by bilateral median position. Such cases cannot yet be explained. Various authors [6] have described similar observations—for instance, the unilateral ligation of the thyroid artery which was followed by bilateral median position of the paralyzed vocal cords. In view of the experimental findings by Hartenau and Schwetz,[15] one must assume that abnormal reflexes with resulting contractures are produced by alterations of the sympathetic innervation. So far, it is certain that the perivascular sympathetic supply assists in the maintainance of the intrinsic muscular tonus.

Laryngoscopic Findings

The complicated and often confused problem of bilateral median immobilization of the cords requires a systematic consideration of our present knowledge. At first, we have to remember the accepted definitions of 1. abductor paralysis, 2. recurrent nerve paralysis, 3. vagus paralysis, and 4. arthritic fixation.

1. As has been sufficiently discussed in the preceding papers,[4-5] *abductor paralysis* is a definite and clearly visible condition. It was well known to the older authors that isolated paralysis of the abducting posterior cricoarytenoid muscle is characterized by the inability of the affected cord to *abduct beyond the intermediate line*. This limited abduction represents nothing but the passive inspiratory relaxation of the normal adductors. What is lost in such a case is the active abducting function of the posticus muscle which normally moves the cord from the intermediate line to the position of full lateral abduction (Stupka [24]). It follows that abductor paralysis has nothing to do with

median position or median fixation of the affected cord. In fact, the cord is not immobilized at all; it is only limited in abduction. The same is true for bilateral abductor paralysis. In this case, both cords fail to abduct beyond the intermediate line, although they can be normally adducted to the midline position of phonation. Since isolated lesions of both abductor branches are vary rare, only a few observers have actually seen this condition. However, it does exist.

Tschiassny [25] goes even further when explaining that the abductor muscles are actually *dilators* of the glottis. He recently discussed the modern concepts of laryngeal paralysis and recalled several well-established observations. In *quiet respiration*, the cords show pendulous motions around the intermediate line. During inspiration, the glottis is wider, primarily owing to the inhibition of the adductor tonus and to the elastic force provided by the conus elasticus. During expiration, the glottis becomes more narrow as the result of increased adductor tonus and inhibition of the posticus muscles. The active function of the dilating abductors comes into play only during *increased* inspiration when the glottis is fully opened to the lateral position of deep inspiration. It follows again that abductor paralysis has nothing to do with midline position.

2. According to the majority of authors, *midline position* of a paralyzed cord is now interpreted as a sign of complete *paralysis of the recurrent nerve*. It is agreed that the paralyzed cord is maintained in this position by the intact function of the external cricothyroid muscle, which is the tensor of the cords (Wagner and Grossmann, cited by Arnold [4]; Stupka [24]; Hofer, cited by Jeschek [16]; Jeschek,[16] and others [4]). For the same reason, the paralyzed cord retains its tonus and tension in midline position (Weleminsky [26]). The same is true in case of bilateral paralysis of the recurrent nerves, which is followed by bilateral median position. Gregg [14] states that the description of this situation as "bilateral abductor paralysis" is a misnomer.

In many cases of median or paramedian position of one or both cords slight *residual movements* can be seen (Luchsinger [18]). During inspiration, the glottis becomes a little wider because the remaining adductor tonus relaxes. In expiration, the glottis is slightly narrowed, owing to the residual expiratory action of the adductors (Stupka [24]). It should be borne in mind that the cricothyroid belongs to the adductor group. Since this external tensor is not affected by recurrent nerve paralysis, its adducting action during expiration will remain intact.

Otherwise, we should remember the tonic innervation via the sympathetic nerve supply as discussed above. Respiratory impulses belong in part to the regulatory mechanism of the vegetative system and must reach the larynx so long as the perivascular supply with sympathetic fibers is functioning. What has been said about residual expiratory adduction is also true with regard to phonation. This function is closely linked to expiration, since its movements are regulated by expiratory patterns.

3. *Vagus paralysis* is the prototype of the complete and combined paralysis of both the superior (cranial) and the inferior (caudal) laryngeal nerve. According to many authors,[4] this type of combined laryngoplegia is characterized by the *intermediate position* of the affected cord. The same is true in cases of bilateral paralysis.

To summarize once more the pertinent observations of many authors, we may follow Tschiassny,[25] who recently surveyed the causes of the median and intermediate positions. As long as the external cricothyroid muscle is not affected, as is normal in case of pure recurrent nerve paralysis, its reflex contracture causes a shift of the paralyzed cord from the intermediate to the median line. This reflex contracture may be relieved by local anesthesia.

Many authors have used this empiric observation for the relief of glottal obstruction by bilateral paralysis of the recurrent nerves with median immobilization. Section of one superior laryngeal nerve, or of one cricothyroid muscle, has repeatedly

been found to cause a shift of the respective cord from the midline to the intermediate line. Tschiassny [25] also analyzed Freedman's recent reevaluation of this procedure.

Unfortunately, the surgical elimination of reflex contracture of the cricothyroid muscle is not always successful. As has been shown by histologic studies, degenerative changes in the joints and ligaments of the arytenoids often develop after laryngeal paralysis. These arthritic changes in the joints produce a *true fixation* of the paralyzed cord. As a result of this fixation, the affected cord is maintained in the midline position even after the cricothyroid has become paralyzed by disuse atrophy, or by inclusion of the superior laryngeal nerve in the paralytic lesion, or by the surgical interruption of this nerve.

This, then, is one exception to the general rule, according to which complete and combined paralysis of both laryngeal nerves is associated with intermediate position of the affected cord. Hence, we must conclude that a paralyzed cord may remain in the median position even after the nerve lesion has progressed to the stage of complete and combined paralysis of both laryngeal nerves.

4. As is well known, the evaluation of paralytic conditions of the larynx requires a test of the *passive mobility* of the affected cord. This is achieved by gently probing the arytenoid after topical anesthesia, as outlined in Jackson's textbook. If the arytenoid can be moved sideways, the cord is found to be *immobilized* as a result of the paralysis. On the other hand, when the arytenoid cannot be moved passively, the position of the cord is due to true arthritic *fixation*. As we have seen, complete paralysis of the recurrent nerve and arthritic ankylosis produce the same result: immobilization in midline (or paramedian) position. The mobility test will then help to differentiate these conditions as follows:

(*a*) *Cord immobilized* in median position with passively movable arytenoid: complete recurrent nerve paralysis with intact cricothyroid function.

(*b*) *Cord fixed* in median position without passive mobility of arytenoid: (1) arthritic ankylosis without evidence of paralysis, (2) arthritic ankylosis in addition to complete recurrent nerve paralysis, or, more likely, (3) arthritic ankylosis in addition to complete and combined paralysis of both laryngeal nerves of long standing.

(*c*) *Cord immobilized* in intermediate position with passively movable arytenoid: complete and combined paralysis of both laryngeal nerves with loss of cricothyroid function.

(*d*) *Cord fixed* in intermediate position without passive mobility of arytenoid: (1) arthritic ankylosis without evidence of paralysis (rare) or (2) arthritic ankylosis in addition to complete and combined paralysis of both laryngeal nerves.

(*e*) *Cord not immobilized,* but with abduction limited at the intermediate line: true abductor paralysis.

Phonetic Signs

It is usually said that bilateral midline immobilization of the paralyzed cords leaves the voice essentially intact. Jackson and Jackson stated, "Usually it is so good as to mislead an inexperienced observer to doubt the presence of a paralysis." They added, however, that the voice may be weakened, slightly impaired in tone, and sometimes hoarse. Similar statements may be found in most textbooks and in many papers (von Bleiweis,[8] Dorendorf,[12] Jeschek,[16] etc.). All observers rightfully stress the severe degree of dyspnea as the predominant complaint.

While these general descriptions are perfectly adequate for the routine practice of otolaryngology, the student of vocal function and, in particular, the phoniatrist are interested in a more detailed analysis. Let us, therefore, discuss the symptoms of paralytic dysphonia as a result of bilateral recurrent nerve paralysis with median immobilization. It will be interesting to compare this vocal disability with the phonic signs of unilateral laryngeal paralysis which were analyzed in the preceding paper.[5]

Again, I recommend a simple method of vocal analysis as outlined before.[3]

1. The *general quality* of the voice may be normal for all practical purposes of the average person. As soon as the highly developed vocal functions are considered, their severe limitation is always noticeable, showing that good compensation of the speaking voice is only partial and limited to simple daily requirements. Most of the patients present a slight degree of dysphonia. This is not an ordinary type of hoarseness (Arnold [1]). It is characterized by a peculiar and easily recognized alteration of the speaking voice which is distorted by a husky, dull, cracked, or nonharmonious quality ("broken pot sound"). Many authors make similar references to an altered, deteriorated, or reduced clarity of the voice (Brahm,[9] de Vido,[10,11] Jeschek,[16] Rieben [22])

Higher degrees of dysphonia with true hoarseness are less common. They can be explained by visible level differences of the two cords, as seen on frontal tomographs. Stroboscopic examination may demonstrate visible differences of vocal cord vibration. Sartorio [23] explained that the variable degree of vocal roughness is correlated to irregularities in the bilateral stroboscopic pattern. It is well known that even a good voice, in such cases, is easily affected by minor influences, such as a cold, fatigue, or nervous aggravation. This shows again that vocal compensation in this condition is labile and incomplete.

2. The *breath volume* used for each phonatory cycle is obviously reduced. Although this factor has not been as thoroughly studied as in the case of unilateral laryngeal paralysis, all observers agree on the severe limitation of phonic respiration. Dorendorf [12] described the voice as breathless, shortwinded, and stridorous. As a result of the reduced vital capacity and of the inspiratory difficulty, the breath volume of each phonation cycle is reduced. This requires an increased rate of respiration during speaking (polypnea). Consequently, the spoken sentences are interrupted by noisy inspirations. Another reason for the waste of air may be the incomplete closure of the glottis when one cord cannot fully reach the midline or when it is in a lowered position. It follows that we must consider several factors of impaired phonic respiration, namely, the limitations of inspiration and expiration, the reduced vital capacity, and the biochemical effects of chronic hypoventilation.

3. *Inspiratory stridor* is the most striking symptom of bilateral median position. Each phonic inspiration is accompanied and disturbed by a moaning sound of variable intensity. This is true "vox inspiratoria," voice produced during inspiration. Because of this inverted mechanism of phonation and owing to the oval shape of the glottis during this phase, the stridor resembles the falsetto voice. Hence, it is not a noise and differs from the irregular and rough stridorous noises which are produced by the irregular and rigid edges of cicatricial types of laryngeal stenosis (traumatic, perichondritic, specific, etc.).

4. *Phonation time* is always considerably shortened. Arnold [1] reported values between three and seven seconds. Similar findings were made by Rieben [22] (5 seconds), Luchsinger [18] (3 to 15 seconds), and Brahm [9] (3 to 12 seconds). This simple test gives information on the efficiency of the pneumophonic sound generator in the larynx. It also demonstrates the general state of the patient's respiratory coordination.

5. *Vocal intensity* is reduced in direct proportion to the amount of air wasted with expiration and not sufficiently inhaled during inspiration. Similar to all types of laryngeal paralysis, vocal intensity remains proportional to the diminished subglottic pressure. Sartorio [23] noticed reduced vocal volume in all seven of his cases. Stroboscopically, he saw reduced amplitude of vocal cord vibration. Apart from these physical reasons, the patients avoid louder intensity because it requires increased inspiratory effort, which tends to aspirate

the cords and thus makes the glottis narrower. This weakness of the voice is regularly noted by all observers (Dorendorf,[12] Rieben,[22] Brahm,[9] Berendes,[6] Arnold[1]). Together with the respiratory effort, the polypnoic air waste, and the general reduction of health, this reduced vocal intensity accounts for the great fatigability of such voices.

6. *Speaking pitch* is less altered than in cases of unilateral laryngeal paralysis. Studying 25 cases, Brahm[9] noticed no major changes, except a slight elevation of pitch in some instances. Dorendorf[12] found that the speaking voice had become higher, sometimes changing to the falsetto register. In my own cases,[1] men were found to speak in the middle register and women used the head voice, both being too high. I also noted a tendency toward abrupt changes between a more resonant chest voice and a weak or breathy higher register. A lowered speaking range was described by Luchsinger.[18]

7. The *vocal range* is always reduced, although to a variable degree. As a rule, the high tones are lost. Among my own reported cases,[1] a man had a residual range of two octaves from E (82 cps) to e^1 (330 cps), having lost the head and falsetto voice. His speaking voice was slightly husky. Two other patients showed a range of D (73 cps) to g (196 cps), respectively, F (87 cps) to g (196 cps), again with marked loss of high tones. In Brahm's[9] 25 cases, one third had a range of one octave or less, one third showed a range between one and one and one-half octaves, while the rest reached between one and one-half and two octaves. Compared with the normal range of two to two and one-half octaves, this indicates an average reduction by one-half to one octave (6 to 12 semitones). Similar opinions were expressed by Berendes,[6,7] Dorendorf,[12] Rieben[22] (one octave), Jeschek,[16] Luchsinger,[18,19] or Sartorio[23] (one octave, usually in the center of the median vocal frequency range, namely between 130 and 520 cps).

8. The *vocal registers* appear narrowed and displaced in relation to the residual vocal range. Their passages are considerably disturbed and displaced. Many times the chest register has lost its full resonance. Head voice and the falsetto register of males are either absent or limited in extension. For the same reason, the middle register may be compressed by the loss of its higher tones. Berendes and Luchsinger[7] described a woman who presented a lower register between c (131 cps) and c^1 (262 cps). This corresponded to an unusually low chest register and sounded clear. A higher register, also with unusual limits, extended from c^1 (262 cps) to b^1 (494 cps). These tones were hoarse. It is generally understood that such higher tones are produced mainly by contraction of the cricothyroid muscle. Since median position of a paralyzed cord is interpreted as a sign of complete paralysis of the recurrent nerve alone, the external tensor and its nerve are assumed to function in such cases.

9. *Special functions* of the voice, such as singing, reciting, and calling aloud, are always lost or severely impaired. Contrary to the slight disturbance of the speaking voice, this limitation is often deplored by the patients. With regard to calling aloud or shouting, it has already been explained that tonal volume is always curtailed.

The deleterious influence of bilateral median position on the singing voice was studied by Jurasz[17] in 1900. Before the onset of paralysis, a male singer had a vocal range of two and one-fourth octaves, from F (87 cps) to a^1 (440 cps). Suffering from tabes, this patient developed bilateral laryngeal paralysis with median position. His range decreased to one octave from B (123 cps) to b or c^1 (262 cps). These tones were not clear and appeared unsuitable for singing. While the patient could perform the "crescendo" increase of tone volume on equal pitch, the "decrescendo" decrease of intensity was no longer possible. As is well known, the tensing action of the external cricothyroid alone is not sufficient for the highly complex process of singing. When

one or both cords are paralyzed, the internal vocalis muscle can no longer perform its specific functions of molding the cords for the various techniques of artistic tone production.

10. *Discrepancies* between the degree of vocal disability and the visible laryngeal appearance are frequently due to poor intralaryngeal compensation. As extensively discussed in the last paper,[5] compensation may be delayed owing to poor motor coordination or as a result of insufficient psychoacoustic talent. One organic reason for marked dysphonia may be the presence of level differences between the two cords (Arnold,[1] de Vido,[10] Luchsinger[18]). This can be verified with laminography.

11. *Functional complications* on an habitual or emotional basis have the same origin as in the case of unilateral paralysis.[5] It cannot be surprising that the awful dyspnea, or a hormonal imbalance following thyroidectomy, should lead to general nervousness or secondary emotional reactions. Such additional factors should be analyzed as explained before.

The Voice After Surgical Laterofixation

As explained at the beginning of this paper, laryngeal paralysis leads to an inverted relationship between the degrees of dysphonia and dyspnea; the wider the glottis, the better is respiration and the poorer is phonation. This unavoidable dilemma dictates careful planning of operations which aim at the permanent widening of the glottis. All surgeons experienced with arytenoidectomy or arypexy agree that such operations improve breathing but impair the voice. It is, therefore, necessary to explain to the patient what he may expect.

It is also necessary to decide what will be of greatest benefit in the individual case. This decision is guided by the main purpose of such operations: to establish good breathing and to conserve a good voice. Obviously, a "good" breathing ability and a "good" voice mean something different to different patients. Manual workers require a sufficiently wide permanent glottis for the performance of strenuous work. Whether or not their voices are hoarse is of less importance. On the other hand, office employees or persons engaged in occupations requiring constant talking need a clear and sufficiently resonant voice for their daily work. Whether or not their breathing may be insufficient for heavy physical exertion is much less important. It follows that manual workers need a *wide respiratory glottis* and do not mind the ensuing dysphonia. Professional speakers need a sufficiently *narrow phonatory glottis* and are less incapacitated by some residual dyspnea.

The effects on the voice of laterofixation have been studied by several authors (Berendes and Luchsinger,[7] de Vido[10,11] and Sartorio[23]). These and other investigators have concluded that the voice is damaged by two postoperative consequences: (1) the lateroposition of one cord and (2) the unavoidable surgical trauma.

(1) The *widening of the glottis* alters the voice in precisely the same manner as any other shift of a paralyzed cord from the median to the paramedian or intermediate position. As a general rule, all those vocal changes are to be expected which were discussed in the previous paper.[5] Accordingly, the preoperative second laryngoscopic stage of median position with slightly dysphonic voice (II. phonetic stage) is transformed to the third laryngoscopic stage of paramedian position with flutter voice (III. phonetic stage), or even to the fourth laryngoscopic stage of intermediate position with severe dysphonia (IV. phonetic stage). De Vido[10] specified that a glottis with an interarytenoid width greater than 5 mm. results in poor voice, obviously because the laterofixed cord is then in intermediate position. With the passing of time the voice improves again because the other cord tends to approach the transposed cord during phonation. For this reason, the surgeon selects the less movable cord for laterofixation.

(2) *Surgical trauma* includes all effects such as postoperative edema, shrinkage of scar tissue, diminished blood supply, and inflammatory infiltration. These influences are magnified by a preexisting rigidity of the thyroid wings (physiologic calcification). Since most of these postoperative sequelae are bound to diminish with time, the voice tends to improve spontaneously. Again, a residual motility of the other cord helps to compensate for the greater width of the glottis, and this cord may learn to cross over the midline to reach the laterofixed cord.

As with the functional results of all other surgical interventions, the effect of laterofixation on the voice is subject to many individual variations. In addition to the general rules as explained above, we also find various types of individual reaction. These are due to variable degrees of psychomotor and psychoacoustic adjustment. Some patients have been found to have a very good voice, while many others complain of vocal deterioration (de Vido,[10] Sartorio,[23] etc.). When analyzing such postoperative vocal disabilities, one may follow the comparison of laryngoscopic and phonetic stages as outlined in the previous paper.[5] Some peculiarities of the voice after laterofixation may be summarized as follows.

Vocal intensity is always reduced for the same reasons as discussed before. The voice sounds weak, breathy, and is easily fatigued.

The *vocal range* is usually narrowed to a few tones. The registers appear more disturbed than before the operation.

The median *speaking pitch* tends to be lowered. This is due, in part, to the postoperative edema and infiltration of the cord. Every increase in mass will lower the frequency of vibration. Otherwise, it has the same reasons as in any case of paramedian or intermediate position.

The *mode of vibration* of the laterofixed cord is altered as seen with the stroboscope. De Vido [10] observed a reduction of vibration to the anterior half of the fixed cord. Being too far away from the other cord, the posterior half of the laterofixed cord could not be induced to passive vibration. Another reason for the asymmetry of vibrations or their unilateral reduction is the edema and infiltration on the operated-on side. The ear perceives these irregularities as vocal roughness or diplophony.

Frequently, the regular preoperative vibrations have been altered to a vertical pattern which always indicates weakness or loss of tension of the affected cord. Sartorio [23] saw the following details. The nonfixed cord had a range of horizontal vibrations from e (165 cps) to f^1 (350 cps) with a maximum around c^1 (262 cps). On the opposite side, the fixed cord performed only vertical vibrations which were often out of phase; hence, the audible roughness of this female patient's voice.

Such changes in the vibratory pattern are easily analyzed with the modern technique of high-speed motion pictures. Berendes and Luchsinger [7] studied a female case and made the following observations. On the side of laterofixation, the phonic vibrations were reduced, showing later onset, smaller amplitude, vertical component, and earlier decay than on the other side.

Individual differences in vocal quality after laterofixation may be due also to *level differences* of the two cords. Often, the surgeon attempts to lower the laterofixed cord in order to obtain an optimal airway. While succeeding in this direction, the level difference achieved can easily produce a dysphonic element of diplophony, flutter voice, or air waste with shortened phonation time.

In such cases, or for whatever other reasons the laryngeal voice generator may be confronted with the task of compensation for the insufficiently closed phonating glottis, the ventricular folds may assume the phonatory function by overadducting and closing over the widened glottis. As in all cases of *vicarious ventricular dysphonia,* this compensatory mechanism may develop in three types: (1) between the laterofixed cord and the opposite ventricular fold, (2)

between the opposite cord and the ventricular fold on the side of operation, or (3) between both ventricular folds. Such ventricular-fold voices vary a great deal in quality.

Summary

This is an analysis of the relationship between phonation and respiration in cases of bilateral median immobilization of the vocal cords. According to modern concepts, this condition results from bilateral complete paralysis of the recurrent nerves.

Differential diagnosis, the influence of sympathetic innervation, and some recent experimental findings are discussed. It is advisable to differentiate true abductor paralysis without any immobilization, paralytic midline position due to recurrent nerve paralysis, arthritic median fixation as a result of arytenoid ankylosis, and intermediate position subsequent to combined and complete paralysis of both laryngeal nerves. Cases of bilateral paralysis follow the same rules as unilateral lesions.

As in the previous paper, the visible laryngoscopic findings are compared with the audible phonetic signs. In general, the details of vocal pathology follow the acoustic laws applicable to an abnormal laryngeal sound generator. Individual deviations can be explained by secondary psychomotor or psychoacoustic interferences with spontaneous intralaryngeal compensation.

Finally, the voice after laterofixation is analyzed according to the main postoperative effects: widening of the glottis and tissue reactions to surgical trauma.

6 E. 79th St. (21).

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Otoplasty

A New Simplified Technique

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Otoplasty for the correction of protruding ears is being performed with increasing frequency, but, as yet, the technique has not been standardized. The purpose of this paper is to present a simple effective method of correcting this congenital defect.

When seen in the newborn, the "lop ear" deformity is easily recognized, and frequently the distraught parents attempt to hold the protruding auricle back with external pressure, but to no avail. As the child grows older, he may become self-conscious and embarrassed by his defect, especially if it is brought to his attention by his family. For this reason, and also because the elasticity of the cartilage in the young lends itself to excellent surgical correction, I believe the preschool age is the opportune time for operation. No alteration in the normal growth of the auricular cartilage is seen postoperatively. Correction may be done at any age, however.

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Anatomy

The pinna consists of two principal cartilaginous portions: the concha, which protrudes at an angle from the skull, and the scaphoid portion, which normally makes almost a right angle with the concha (Fig. 1*A*). This angle is known as the antihelix. It is the incomplete formation of this antihelix fold-back that causes the scaphoid to protrude and forms the "lop ear" defect. According to Foman,[1] a "lop ear" deformity is said to exist if the scaphoid protrudes from the side of the head at an angle greater than 30 degrees.

Surgical Considerations

In general, there have been three methods of surgical correction for protruding ears. In the early stages of the operation, a wedge of skin was removed from the pos-

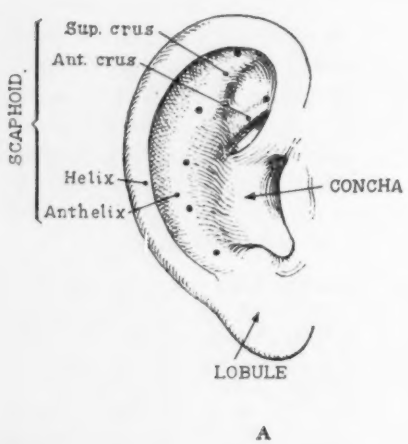


Fig. 1. — *A*, anterior surface of pinna, showing antihelix with superior and anterior crura marked with dye. *B*, posterior view of antihelix, with dye marks and approximate area of skin incision.

Fig. 2.—Typical sharp antihelix formation after removal of cartilage from antihelix by old technique.

terior pinna in the mastoid area. The saucer-shaped pinna was sutured back against the skull, and one defect merely was substituted for another.

The next technique, nicely described by Leonardo,[2] created an antihelix by excision of cartilaginous wedges along the conchal scaphoid junction and folding the scaphoid back by posterior perichondral sutures and skin sutures. Although this created an antihelix fold, many operators felt that it produced a sharp angulation on the anterior surface of the pinna (Fig. 2). To obviate this defect, Erich[3] developed a technique whereby a gauze roll was sutured to the posterior surface of the pinna by a double row of penetrating sutures through the full thickness of the external ear. This method created a more complicated postoperative technique and also subjected the patient to the hazards of penetrating cartilaginous sutures. Others have used a wire brush to weaken the tension of the pinnal cartilage and thus create a gently curving antihelix.

In the past four years, I have had the opportunity of using the simpler method about to be described and observing the postoperative results. This technique breaks the spring in the auricular cartilage by a series of parallel incisions through posterior perichondrium and cartilage and without excision of cartilage, except in rare cases. The pinna is held back in normal position with permanent white nonabsorbable surgical (silk) box type perichondral sutures.

Technique

The deformed auricle is folded back into correct position, and a series of perforations are made with a long hypodermic needle from the front surface, delineating the antihelix on the skin of the posterior pinna by touching the side of the needle with brilliant green or gentian violet and then withdrawing the needle (Fig. 1*B*). An elliptical wedge of skin is removed from the posterior pinna, varying at its greatest central width from 1 to 3 cm. in size, with the use of the antihelix with its superior crus extension as the midpoint of the segment. Care should be exercised to avoid going up to the very top of the ear, as the suture line in this area might be visible postoperatively in keloid-formers. At the lower pole of the wedge, or in the region of the lobule, the amount of skin removed depends on the findings present. Should the lobule be turned forward, the skin removed should be enough to pull the lobule, which contains no cartilage, back into its normal position at the time of closure. In other areas, the perichondral sutures hold the auricle in its corrected relationship.

The skin adjacent to the excised area is undermined for about 1 cm. to facilitate later closure (Fig. 3*A*). The antihelix and superior crus are

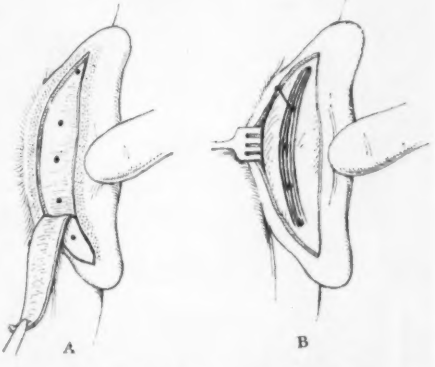


Fig. 3.—*A*, excision of postauricular skin. Stippled area shows undermined skin to facilitate later closure. *B*, vertical parallel incisions through cartilage to break tension. Also, single bold incision to form anterior crurus.

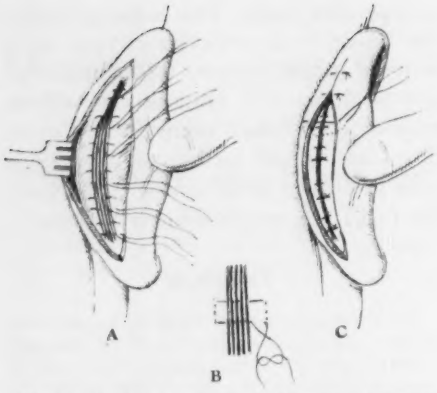


Fig. 4.—*A* and *B*, permanent box type mattress sutures of 0000 white silk (Deknatel) inserted and tied alternately from above and below. *C*, closure of skin with black silk.

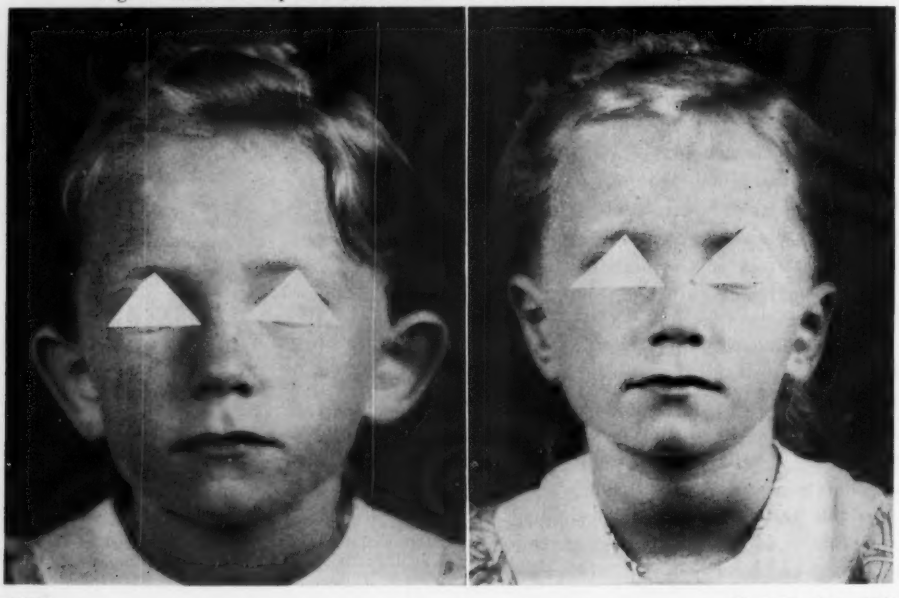
then formed by a series of about six closely parallel linear striations through the posterior perichondrium and auricular cartilage, centering on the dye points previously created (Fig. 3*B*). The striations should end about 1 cm. from the upper margin of the upper ear to prevent unnatural severe angulation in this area. However, the striations should be sufficient to allow the ear to be held back in normal position with very little tension. The anterior or inferior crus is formed by making one bold incision along the points previously marked on the anterior ear.

About six box type mattress sutures of 0000 white silk are inserted through the perichondrium as shown in the diagram (Fig. 4*A* and *B*). These are not tied until later. Care should be used to pass these sutures subperichondrally through the area of vertical incisions in order to create a gently curving antihelix fold or tube. The pinna should be held back in its normal position by the assistant and the sutures tied alternately from above and below. For added strength I usually insert two or three retention sutures outside of the main suture row. The skin is then closed by any satisfactory type of interrupted everting suture with use of 0000 black silk.

With careful hemostasis I have avoided the use of drains. Saline-moistened cotton is applied to the anterior and posterior sides of the pinna. A mastoid type pressure dressing is applied, with the use of caution to apply posterior pressure to each ear. Bilateral correction, when needed, is routinely performed at the same procedure, since less than one hour is required for each side. The ear dressing is not disturbed for 10 days, and the patient is usually released from the hospital on antibiotics 24 hours after surgery.

The dressing and sutures are removed on the tenth postoperative day at which time two or three Band-Aid type strips are applied from the pinna to the mastoid skin in order to prevent tension on the operative area during sleep or trauma. These are removed four days later, and the patient is discharged.

Fig. 5.—Bilateral "lop ear" deformities before and after correction, anterior view.



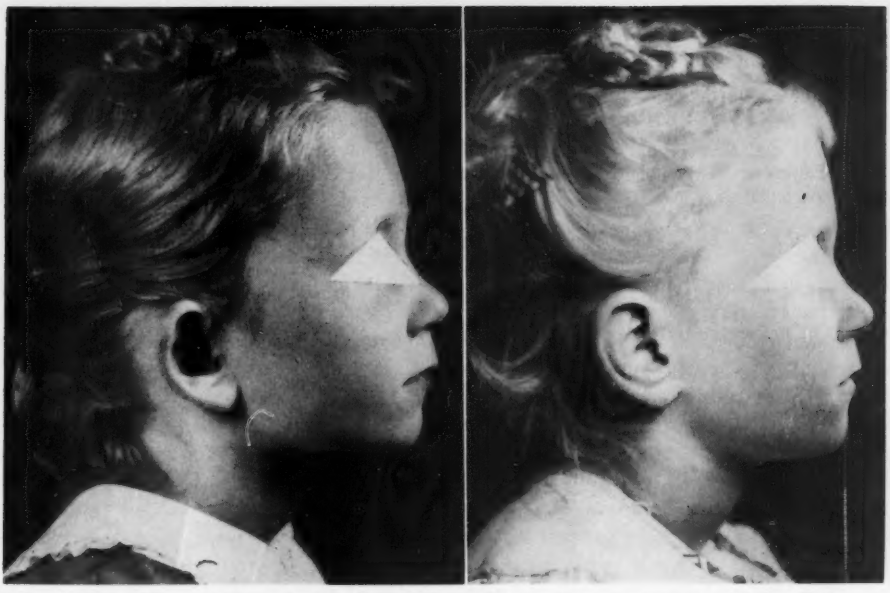


Fig. 6.—Same as Figure 5, right lateral view. Note gentle curvature of antihelix. Postoperatively, by technique described in text. (No cartilage was excised in this case.)

Intratracheal general anesthesia is used routinely, but a small amount of procaine hydrochloride (Novocain) and epinephrine may be injected postauricularly to reduce vascular ooze. Postoperative discomfort is surprisingly slight and usually limited to head-dress pressure.

Comment

In occasional ears, the concha may be particularly wide, positioning the corrected scaphoid too far from the side of the head. This can be eliminated by making the vertical cartilaginous striations which form the antihelix more medial than usual or on the conchal side of the dye markings previously created. If this is not adequate to correct the excessively wide concha, a wedge of cartilage may be removed from the concha just medial to the striations. It should be noted that any cartilage removed is not from the dome of the antihelix and, as such, will not affect the final shape of this structure. Occasionally in the region just above (cephalad to) the lobule there is a double layer of cartilage where embryologically the conchal and scaphoid cartilages overlap. A small wedge of this excess cartilage may be removed or cross hatched to eliminate any abnormal prominence in this area.

Frequently, one ear may actually be larger than the other and this may render successful correction more difficult. In the young, the external ear may be disproportionately large. This discrepancy is usually corrected with normal growth and should not influence our therapy in "lop ear" deformities. Actually, the correction of the ear deformity often reduces the illusion of an enlarged pinna.

Summary

A simple method for the correction of protruding external ears is presented. It usually avoids the excision of cartilage, which may cause sharp unnatural angulations on the anterior pinna. It simply creates the normal antihelix fold by a series of parallel cartilaginous striations and permanent perichondral sutures. This method makes for most simple postoperative care. It gives very satisfactory results, while re-

ducing the risk of postoperative complications and failure.

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Experimental Intralaryngeal Injection of Radioisotopes

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This report is concerned with the experimental injection of $CrP^{32}O_4$ and $Cr^{51}P^{32}O_4$ (radioactive isotopes) into the submucosal spaces of the larynx. Observations were made of the local effects of the radiation from P^{32} on the larynx and the distribution of the $CrP^{32}O_4$ within this organ. These observations have expanded our knowledge of the anatomy and physiology of the larynx and have proven helpful in understanding the natural history of certain diseases of the larynx.

The ability of the normal larynx, as a whole, and of its component parts to tolerate injected radioactive material has been observed and the way opened for a clinical trial of radioisotopes in the treatment of certain cancers of the vocal cords. It has likewise been established that if radioisotopes are to be used for the treatment of cancer of the larynx the injection needs to be made in accordance with anatomical details which have been observed in the course of these experiments.

Studies of spread and distribution of injected radioisotopes have supplemented observations made with the use of a colloidal dye (4% Direct Sky Blue, Wyeth)[1] and have provided further identification of submucosal compartments of clinical importance, originally described by Hajek, in 1891,[2] and elaborated upon in our previous reports.[1,3] The pattern of lymphatic flow, ordinarily considered to be as illustrated in Figure 1, has been further identified in the experimental animal and in observations upon humans.

Radioactive chromic phosphate is useful in these anatomical observations by reason of its being identifiable over longer periods of time than dyes or radiopaque contrast media. Observations within the larynx by the latter methods are possible for only a few hours or days, at the most, but by the use of radioisotopes, the period of observation has been extended in our experiments for as long as eight weeks, which by no means exhausts the time limit of usefulness.

The $CrP^{32}O_4$ was prepared by a modification of the method of Morton.[4] The particle size was determined by electronmicroscopy and was found to vary from 0.25μ to 0.5μ. The final solution for injection was prepared by suspending the particulate $CrP^{32}O_4$ in a suspending media containing 10% by volume of hyaluronidase (Wydase) and 1:100,000 epinephrine. A small amount of Evans blue dye was added as a visual aid to detect leakage from the injection sites and to observe its spread within the laryngeal tissue. $Cr^{51}P^{32}O_4$ was prepared in a similar manner. Cr^{51} was employed in these experiments so that its γ-emission might be utilized for external scanning of the larynx and the cervical region post injection.

In order to properly introduce radioisotopes into the submucosal structures of the larynx, a special automatic injector is required (Fig. 1) capable of delivering exact amounts of the colloid. The Gamble-Libby[5] unit is excellent for this purpose and has been used in these experiments. This is an electromechanical leak-proof injector, activated by a foot switch, capable of delivering fixed amounts of fluids through a 3 to 5 ft. section of flexible tubing into a

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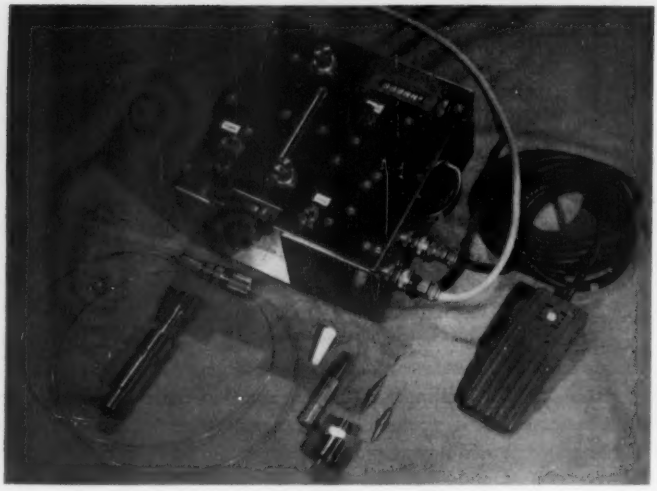

Fig. 1.—The Gamble-Libby automatic injector.

needle, the orifice of which is located a few millimeters proximal to the tip. Specially prepared curved needles with a 60- to 90-degree offset have proven most practical. The amount of fluid delivered by the automatic injector can be varied from 0.065 to 0.4 ml. For these experiments, the 0.065 ml. injection volume was used.

Technique of Study

Studies were carried out upon human cadaver larynges, living dogs, living pigs, and living humans. Injection of dyes preliminary to the use of radioisotopes was done into cadaver larynges at various sites, and gross observations were made of the limit of spread taking place prior to rupture of the anatomical structures from pressure of the injected fluid.

Two humans, prior to laryngectomy, each having unilateral carcinoma of such limited extent as not to interfere with the normal anatomy, were selected for observation. Dye 0.5 ml. was injected through a direct laryngoscope in several fractions in and about the true vocal cord of the uninvolved side, and the laryngectomy was completed four hours later. Gross observations of the spread of the dye were made. The experiment was repeated in living dogs and pigs, with the use of dyes for preliminary study and radioisotopes for longer-term observations. Experiments were conducted both with the larynx split in the midline anteriorly and with the larynx intact.

The dogs used in these experiments were prepared under general anesthesia and with sterile surgical techniques. The interior of the larynx was exposed through an anterior midline incision of the thyroid cartilage. The various compartments of the larynx were identified as indicated

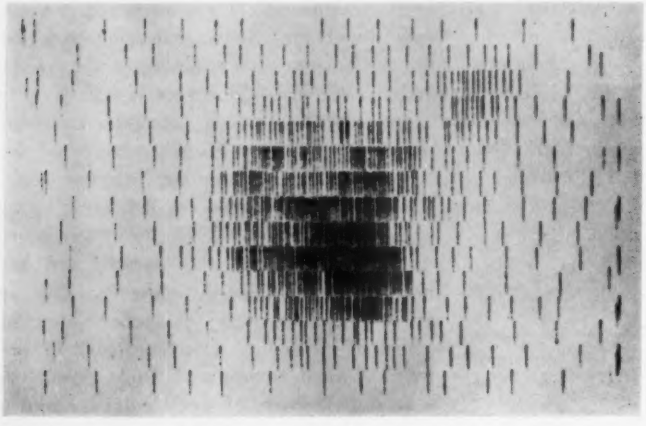

Fig. 2.—Scintigram after a bilateral diffuse injection of radioisotopes throughout the larynx. Note cervical node which has taken up radioactive substance (upper right).

in Figure 2. The Gamble-Libby automatic injector, Figure 1, was employed to deliver 0.065 ml. of the radioactive material to each of 8 to 10 injections to one or both sides of the exposed larynx. In these experiments, the amount of radioactivity varied from 25μc. to 70μc. per injection.

Scintigrams of all larynges of dogs given injections of doubly labeled $Cr^{51}P^{32}O_4$ were made at selected periods up to eight weeks. The dogs were killed, and the laryngeograms were superimposed upon the larynx of each of them and photographed. Micro- and gross radioautographs of the larynges and cervical nodes were made subsequent to autopsy.

Gross observations after the injection of dye into the larynx were made of the associated lymphatics to determine the spread of the dye by lymphatic channels. When radioisotopes were used, cell counts were made of samples of blood and of all organs, including the adjacent and distant lymphatics, to determine the degree of radioactivity present. Excreta were examined to determine the loss of radioactive substance by these channels.

Anatomical Observations

A. Relationship Between Opposite Sides. If $CrP^{32}O_4$ (radioactive isotopes) or colloidal dyes are injected into one side of the larynx at any point above the lower level of the cricoid cartilage, the spread of the material by the lymphatics will be such that there is, with exceptions to be described, no crossover to the opposite side. This conforms to the pattern of lymphatic distribution, the intralaryngeal lymph channels forming collecting lymph vessels which converge, ascending parallel to each other alongside the midline. Observations concerned extended to 12 days in the instance of the dye, after which it could not be any longer identified, and to 8 weeks in the instance of the radioisotopes, representing the longest period over which observations have been made. It is not believed that the degree of isolation of one side from the other has previously been demonstrated.

Two exceptions are noted. In the instance of pigs, dye injected submucosally 1.0 cm. below the level of the vocal cord passes upward, fails to enter the margin of the vocal cord, and reappears on its superior surface. The dye stained the wall of the saccule and crossed the midline at the anterior commissure to spread as a thin band along the superior aspect of the opposite cord. The second exception was observed in the cadaver larynx of dogs. Dye forcibly injected into the ventricle appears in two lymphatic channels, one immediately above and the other immediately below the ventricle. These converge anteriorly, forming a single channel which crosses the anterior commissure. The channel then splits into two, surrounding the ventricle on the uninjected side. No crossover to the isolated side was noted at any other level, including injection of the epiglottis (Addendum).

In the living human, prior to laryngectomy, dye injected in and above the true vocal cord spreads diffusely, staining the entire homolateral side including the arytenoid cartilage and extending to its posterior surface to enter the pyriform sinus area. No crossover beyond the midline occurs at any point, the midline itself being sharply delineated. The lower limit of spread is at the lower border of the cricoid cartilage. This is likewise sharply delineated, representing the separation of the lymphatic bed of the larynx from that of the trachea. This is likewise of the greatest practical importance. This finding is discussed in detail.

Since conclusions concerning the spread of dye depend upon visual observation alone and there is no opportunity to learn of its ultimate fate or its area of dissemination prior to death, this method of observation can be considered only as a preliminary study requiring the use of radioisotopes for confirmation and for observation of details.

Corroborative observations upon possible crossover beyond the midline after an extended period of time were made upon living dogs with use of radioactive isotopes. Three dogs were used in similar experiments, and the findings were alike. Ten submucosal injections of 0.065 ml. containing 37μc. of $Cr^{51}P^{32}O_4$ were made on the right side of each larynx. The injections were made at various sites throughout the larynx, including the subglottic area, the free margin of the vocal cord, the ventricle,

the aryepiglottic folds, and the epiglottis. With the injector needle vertically placed, one injection was made in each area just lateral to the midline. The dogs were killed eight weeks later. No demonstrable cross-over beyond the midline was found by use of the scintiscanner. The side where the injection was given, however, remained markedly radioactive. The lymph nodes of the neck were scanned, radioactivity being demonstrable only on the side of the injection, while nodes on the opposite side remained free of radioactivity.

B. Vocal Cord Bursa.—Preliminary observations after the injection of dye below the level of the cord indicate that the dye passes upward, staining the internal surface of the saccule, but after five hours fails to enter the lymphatics of the free margin of the vocal cord—this area representing the vocal cord bursa, which remains free of dye. These preliminary findings were augmented by experiments with use of radioisotopes. Ten injections of 37 μc. each (0.065 ml. per injection) were made 0.5 cm. below the level of the vocal cords. Eight weeks later, the dogs were killed and radioautographs were made of the entire larynx of each. The findings obtained by dye studies were confirmed, no evidence of radioactivity being demonstrable in the area corresponding to the level of the vocal cords (Fig. 3).

The vocal cord bursa, therefore, from the standpoint of lymphatic distribution as

Fig. 3.—Radioautograph of larynx after an injection of $CrP^{32}O_4$.

demonstrated by the injection of radioisotopes as well as dye, is isolated from the remainder of the larynx. With dye studies, this applies to the human cadaver larynx as well as to the living dog. The reverse situation, however, does not invariably apply. It has been noted that dyes injected within the bursa, particularly in dogs, sometimes enter the saccule, distending it and staining it, but this does not occur in every instance. Studies, now under way, of the fate of radioisotopes injected directly into the laryngeal bursa are expected to clarify this point but have not yet been completed.

C. Lower Limit of Distribution of Laryngeal Lymphatics.—Determination of the lower limit of spread of the laryngeal lymphatic plexus is of the greatest practical importance and, in large measure, needs to be considered the limiting factor in performing certain partial resections of the larynx. Additional experiments have, therefore, been carried out in an attempt to establish this lower level with certainty. These consist of observations after the injection of doses of radioactive isotopes in sufficient strength to produce widespread necrosis. The level of radionecrosis is invariably sharply demarcated at the lower level of the cricoid cartilage, amounting in some instances to almost complete destruction above this level, with normal trachea wall remaining below (Fig. 4). It is apparent, therefore, that radioisotopes introduced into the lymphatic circulation of the larynx do not extend below the lower limit of the cricoid cartilage, corresponding with the previously described separation of laryngeal lymphatic channels above this level from the tracheal lymphatic circulation immediately below. This finding, representing the isolation of the larynx above from the trachea below, has been confirmed visually by the use of dye in the living human and in the living dog. The former has been mentioned parenthetically in the paragraph having to do with the ipsolateral distribution of injected dye. When dye is injected in and above the vocal cord in the human four hours prior to laryngectomy, it fails to cross to the opposite side and a sharp line of demarcation occurs at the lower base of the cricoid cartilage. This observation has been repeated in dogs. Dyes injected in minute amounts between the level of the vocal cord and the lower border of the larynx spread upward and downward on the side where the injection has been given, the latter distribution terminating abruptly in a straight line at the lower border of the cricoid cartilage. This finding, as evidenced by all methods of observation, is rather startling in that one could consider the larynx and trachea to be a continuous tube with free communication of lymphatics between them, but such, evidently, is not the case.

Fig. 4.—Necrosis of interior of larynx 36 days after injection of 50μc. of $CrP^{32}O_4$ per each of 20 injections. Ulceration is limited inferiorly to lower border of cricoid cartilage, as is always the case.

Anatomical Factors Influencing Radionecrosis After Injection of Radioisotopes

At least two factors contribute to the increased sensitivity of certain areas of the interior of the larynx to radionecrosis after the injection of $CrP^{32}O_4$. One is the close adaptation of the mucosa to the cricoid cartilage in the subglottic area and the other the confluence of lymph channels at the mid-

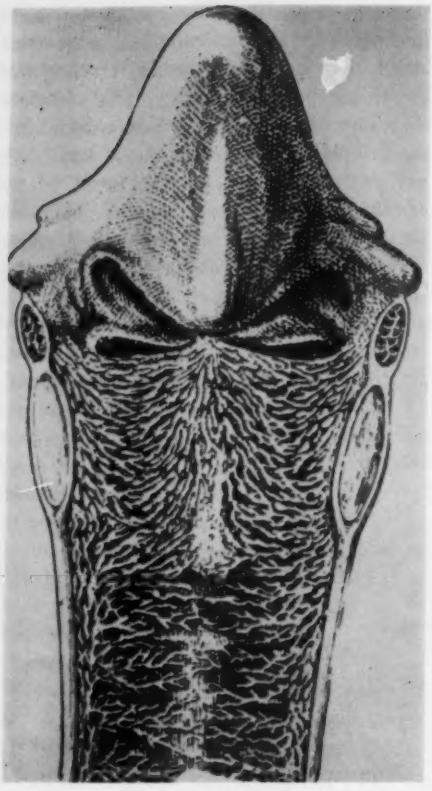

Fig. 5.—Schematic representation of lymphatic of interior of larynx with convergence of lymph channels at the midline near the upper level of posterior commissure.

line near the upper level of the posterior commissure (Fig. 5). If radionecrosis does occur, it is most apt to be in the mucosa closely adapted to the cartilage particularly near the midline. The point of greatest susceptibility seems to lie posteriorly just below the posterior commissure, at which site small ulcerations are frequently found. This corresponds to the confluence of a greater number of lymphatic channels than is to be found at any other site (Fig. 5). Radionecrosis is least apt to be found overlying the intrinsic muscles of the larynx and is seldom seen in the ventricle or aryepiglottic folds. With massive dosage, however, no area, of course, is immune.

Fate of Injected $Cr^{51}P^{32}O_4$ and $CrP^{32}O_4$

The clinical experience of Mumma [6] with cancer of the oral cavity and animal experience [7] indicate that radioactive chromium phosphate, prepared as described and injected into muscle or lymph nodes, remains at the local site of injection in sufficient quantities to be cancerocidal. When injected into mucosal or submucosal areas, it is distributed to the regional lymph node bearing areas. The interior of the larynx, with its paucity of muscular substance and its semicompartmentalization, affords a unique opportunity to study the effects of and the fate of certain colloidal or particulate radioactive materials when these are injected into its various compartments.

In a series of 10 dogs the following redistribution and losses of $CrP^{32}O_4$ were found.

1. A small amount of $CrP^{32}O_4$ escapes into the venous circulation at the time of injection. This was observed by examining the blood for radioctivity. This small amount of initial radioactivity is rapidly removed by by the liver and spleen.

2. Due to the nature of the structure of the laryngeal tissue, it was found that 22.8% $\pm$6.0% of the injected dose was lost by oozing from the injection site. This was determined by measuring the radioactivity collected on sponges.

3. During the first 24 hours after injection, it was found that 20.9% $\pm$6.5% of the initial radioactivity in the neck was lost to other organs in the body.

4. Over a period of from one day to two weeks after the injection, about 21% of the radioactivity found in the larynx at 24 hours is redistributed into the regional lymph nodes.

5. Thus, of the original calculated dose, only approximately 50% remains at the sites of injection within the interior of the larynx because of the above losses.

6. The distribution of the $CrP^{32}O_4$ that is lost from the sites of injection (Paragraph 3, above) was studied in 11 dogs. It was found that the radioactivity in the

liver accounted for from 0.0% to 8.2% of the loss; the spleen, 0.0% to 2.2%; the intestines, 0.0% to 6.0% *; the stomach, none; the kidney, 0.0% to 1.3%, and the lungs, 0.0% to 0.05%. Counts of the feces and urine showed little or no radioactivity.

By this technique, it has been possible to deliver to the interior of the larynx from an average of 13,380 to 27,230 rep per gram of tissue when utilizing 25μc. or 50μc. of $CrP^{32}O_4$ per each of the 18 to 20 injections into the mucosal areas of the interior of the larynx of the dog.

Clinical Implications

One cannot fail to be impressed with the clinical implications of this study. The isolation of one-half of the larynx from the other (except by the demonstrated crossover of lymphatics at the ventricles) corresponds to the known behavior of many cancers of the larynx in which enormous tumors occupy one side of the larynx without involvement of the other. Similarly, there appears to be very great resistance to the extension of laryngeal tumors from above to the trachea below, corresponding to the sharp delineation between the two as demonstrated in the destruction of the larynx by radioisotopes. Further detailed studies, not herein described, demonstrate that certain laryngeal compartments identifiable by dye and radioisotope studies correspond to the observed behavior of spread of many intralaryngeal neoplasms. These findings will be reported upon in subsequent communications.

The importance of bearing in mind observations reported herein as they apply to the surgery of the larynx can hardly be overestimated. Surgical procedures for cancer need to consider the likelihood of routes of spread of these tumors, in which the anatomical details of lymphatic flow are of the foremost importance. With these observations in mind, one can almost venture to predict the route of spread of these lesions and can be guided accordingly in deciding upon the technical surgical procedures to be followed. A subsequent communication will present the details of this correlation, the anatomical details, and the surgical indications.

It has likewise been established by these observations after experimental intralaryngeal injection of radioisotopes that the latter remain within specific areas of the larynx long enough and in sufficient dosage to be considered of therapeutic value and that methods for the injection of radioactive isotopes into the larynx are feasible and practical.

A further report on the tolerance of the larynx to varying doses of radioactive isotopes is in preparation.

Department of Surgery, UCLA Medical Center (24).

Addendum

Attempts have been made to repeat this observation in humans and in the dog. It has not been possible in any subsequent experiment to demonstrate these lymphatic pathways. Attempts to observe them by the injection of dyes in the living dog have likewise been unsuccessful.

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* This may be the result of the ingestion of material coughed up from the larynx and swallowed.

Anaplastic Carcinoma of the Larynx Occurring in a Young Woman

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Carcinoma of the larynx is predominately a disease of males and is most frequently seen in the older age groups. Weinstein,[1] in his case report, states that the average age of persons with disease is 60, and 92% of them are men. He reported a case of well differentiated squamous-cell carcinoma in a Negro woman, age 19, treated successfully by complete laryngectomy. In the series of 93 cases reported by Negus,[2] 90 were men, although his youngest patient was a woman, age 26. In the series reported by Martin,[3] the youngest patient was a woman of 25. Other reports of laryngeal carcinoma are those of Lejeune and Lynch,[4] age 20; Orton,[5] age 12½; Walsh and Beamer,[6] ages 12 and 13, and Jackson and Jackson,[7] age 3 years. These cases, except that of Orton, showed no evidence of distant metastasis. In his case, metastases to the knee with death from the tumor occurred in about nine months.

The case to be reported is that of a highly malignant oat-like-cell carcinoma occurring in the larynx of a 25-year-old woman shortly after a full-term delivery. There is also a history of x-ray treatment to the tissues of the neck for a tuberculous lymphadenitis.

Report of a Case

A 25-year-old white woman was first admitted to Huron Road Hospital on Aug. 14, 1953, for delivery after a normal, full-term pregnancy. She was again admitted to this hospital on June 26, 1956, when she was again at full term. A normal child was delivered. At this time, the patient stated that she had had a sore throat and some difficulty in swallowing for several months prior to admission. A review of the past history revealed that she had spent three years in a concentration camp in Russia. After this, she was hospitalized in Germany for an entire year for the treatment of malnutrition. She developed tuberculous adenitis in the cervical region while confined and was treated by bed rest, incision, and drainage of the involved lymph nodes. She had had no other significant illnesses. The family history revealed that her mother died of pulmonary tuberculosis and one sister was living with active pulmonary tuberculosis.

Physical examination five days after her second full-term delivery revealed the patient to be well nourished, well developed, cooperative, and mentally alert. There was no enlargement of the thyroid gland. There were several scars in the cervical region, particularly in the anterior cervical chain, which were the result of incision and drainage of the tuberculous lymph nodes. There were some hard, shotty areas palpable in this area which were thought to represent partially calcified old inactive tuberculous adenopathy. The trachea was in the midline. The heart and circulation were not remarkable. The blood pressure was 120/80. The lung fields were clear. There were no other abnormalities. The basal metabolic rate determined at this time was +7.

A chest x-ray showed both leaves of the diaphragm to be normal in position and contour. The heart and great vessels were normal in size and configuration. There were no abnormal mediastinal masses, and there was no evidence of pulmonary or pleural pathology.

Examination of the esophagus fluoroscopically and radiographically with spot films showed the midportion of the esophagus at the level of the hilus of the lung to present two small out-pouchings, having the appearance of two ulcerative lesions on the right anterior aspect of the esophagus. It was thought that these areas could represent early diverticulae or possibly ulceration from extension of tuberculous lymph nodes.

Esophagoscopy revealed the ostia of two small diverticulae in the midportion of the esophagus. There was no other significant abnormality noted.

Examination of the thoracic and lumbosacral spine demonstrated a defect in the arch of the

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fourth lumbar vertebra which was thought to be congenital.

The patient had a strong psychosomatic component, and it was felt that her most likely difficulty at this time was functional in nature and that there was no organic basis for her difficulty in swallowing. She was accordingly discharged on June 29, 1956.

She was next seen in December, when she complained of continued difficulty in swallowing and an abnormal sensation in the throat. The patient would not consent to be admitted to the hospital for evaluation, and it was necessary to study her on an ambulatory basis. There was no change in the physical findings over those mentioned in her previous hospital confinement. A biopsy of one of the cervical lymph nodes was carried out to ascertain whether or not there was an active process in this area. At this time bronchoscopy and indirect laryngoscopy were also performed. No abnormality was noted in the larynx. The vocal cords moved freely on phonation, and the structure of the larynx was completely normal. A 9-40 bronchoscope was inserted. The trachea was in the midline, and no abnormality was noted at any point from the level of the larynx down to the carina. The carina was sharp and movable. The right and left main stem bronchi were not deviated. All the major segmental orifices on both sides were visualized, and there was no evidence of abnormality. The bronchoscope was removed and the patient allowed to leave the operating room in good condition.

Histologic examination of the excised lymph-node was compatible with a diagnosis of inactive tuberculous adenitis.

The patient was next seen in April, 1957. She was admitted to Huron Road Hospital on April 17, 1957, with a chief complaint of "shortness of breath, a chronic cough, and weight loss." At this time, she stated that since December, 1956, at which time she had been subjected to bronchoscopy, she had had progressive shortness of breath. This was severe on minimal exertion. She also complained of difficulty in breathing when lying down. She had experienced chest pain which was usually retrosternal. She developed a rather severe, hacking, nonproductive cough which was not relieved by change of position or resting. Shortly before her admission, the cough became almost continuous. There was no hemoptysis. In the two months prior to admission, she noticed some change in the tone of her voice and had a tingling sensation in her throat. Associated with this was anorexia and weight loss of approximately 15 lb.

On this examination, she gave the additional information that after her tuberculous lymph nodes were incised and drained she was given a rather extensive course of x-ray therapy while in Germany, in 1943.

At the time of this admission, the patient appeared to have an entirely different symptomatology than that which she had presented formerly.

On physical examination she obviously had difficulty in breathing and a marked inspiratory stridor. She showed evidence of recent weight loss and appeared chronically ill. She remained alert and cooperative, however. Examination of the cervical region disclosed no change over that of the previous examination, and the remainder of the physical examination was not significantly different.

Routine laboratory data were as follows: Hgb. 13.2 gm., 85.2%; WBC 6,100, with 71% polymorphonuclear cells, 28% lymphocytes, and 1% monocyte. Urinalysis: specific gravity 1.012, albumin 2+, sugar and acetone negative. Serology negative. Sedimentation rate 51 mm. per hour. Fasting blood sugar 93 mg/100 cc. of blood. Blood urea nitrogen 14 mg/100 cc. of blood. Sputum for acid-fast bacilli negative on direct smear and culture.

On April 16, 1957, she was taken to the operating room, where a laryngoscopy was done. The findings were as follows: The epiglottis was normal in appearance. The arytenoid cartilages and entrance to the larynx appeared to be within normal limits. Directly below the vocal cords, which moved with some limitation, there was narrowing of the larynx. A 6-35 bronchoscope was then passed into the larynx and an attempt made to pass it into the trachea. The obstruction in this area to the larynx, just inferior to the vocal cords, was so pronounced that it would not permit passage of this instrument. There appeared to be a circumferential narrowing, and the tissue was very firm and not nodular at any point. A biopsy was done of the tissue in this area with some difficulty because of its hardness. The bronchoscope was withdrawn, as at this time she became markedly cyanotic. A tracheotomy was carried out immediately and overcame the obstructive element present. The tissue was submitted to the Department of Pathology, and a diagnosis of neoplastic tissue, type undetermined, was returned. The biopsy specimen was very small and it was suggested that another be obtained. Accordingly, this was done in a few days, and a diagnosis of small-cell carcinoma (Fig. 1) of the larynx made from this specimen. The patient was treated with x-ray therapy, with three portals being attacked, the anterior, right, and left cervical regions, with a total of 1,500 r being applied to each portal. The patient improved moderately and lost most of her symptomatology and was discharged on May 18, 1957, in fair condition. She did not tolerate removal of the tracheotomy tube.

She was next admitted to Huron Road Hospital, on July 31, 1957, at which time she complained of pain in the vertebral column. This had been present for several months and had become increasingly

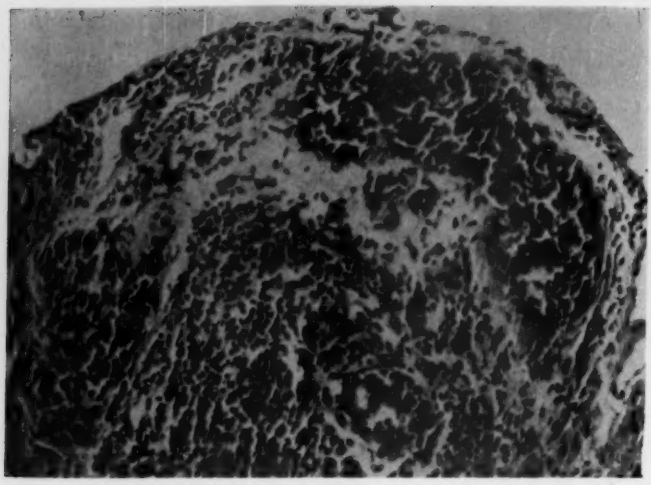


Fig. 1.—Original biopsy specimen, showing small spindle-cell type of tumor cells in larynx.

severer. She had also developed a recurrent cough and some insomnia.

On physical examination, there was no significant change over her previous hospital admission.

X-ray studies of the chest revealed the evidence of marked pulmonary metastases scattered homogeneously throughout both lung fields. Lateral views of the lumbar spine, pelvis, and sacrum showed no evidence of bone metastases at this time. A lateral view of the skull also failed to reveal any bone metastases. She was given 5 mg. of mechlorethamine hydrochloride (nitrogen mustard) daily for four days and discharged in unimproved condition on Aug. 24, 1957.

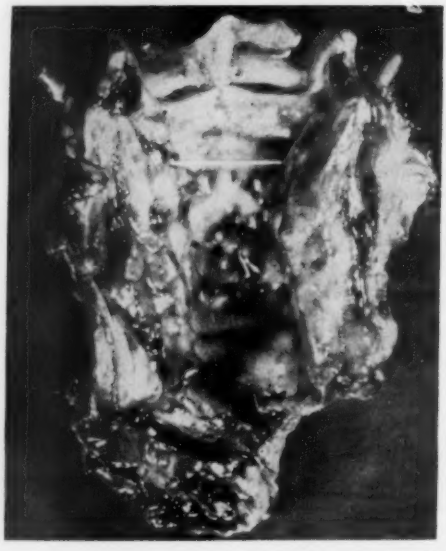
She was readmitted on Sept. 25, 1957 in obvious terminal condition. She complained of severe chest pain, pain in the vertebral column, difficulty in breathing, frequent paroxysms of coughing, and marked weight loss.

Examination of the bony pelvis showed osteoblastic metastases involving both iliac bones and involvement of the right femoral neck. The lung fields were the site of extensive metastases.

The patient was treated by palliation until her death, on Nov. 9, 1957.

Autopsy examination was carried out a few hours after death. When the thorax was opened there was no evidence of free fluid in either pleural space. Scattered throughout the parietal pleura were numerous nodular masses having the appearance of neoplasm. Several similar areas of neoplastic tissue were seen in many ribs, the largest of which was in the anterior portion of the left seventh rib. The respiratory tract was removed, including the larynx. The larynx showed the superior portion to be completely normal, with the true and false vocal cords intact. Just inferior to the true vocal cords on the anterior surface of the larynx was an area of ulceration surrounded by neoplastic tissue (Fig. 2). This tumor was fleshy, somewhat soft, and pale yellowish-gray and laterally extended to the tissue around the thyroid and cricoid cartilages, forming a mass approximately 4 cm. in length and 1 cm. in thickness. The remaining trachea showed nothing of significance except for the stoma, which had been made for the tracheostomy. The lungs were considerably increased in volume, and the pleura was thickened. There were numerous small subpleural tumor deposits bilaterally. On section, the lungs showed diffuse neoplastic

Fig. 2.—Gross specimen of carcinoma of larynx removed at autopsy.



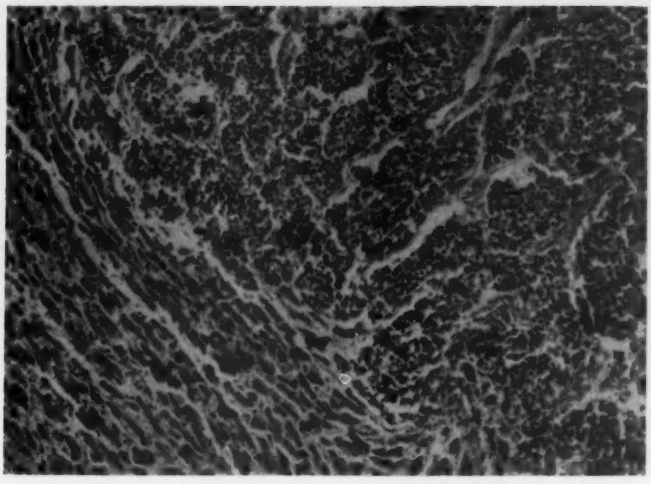


Fig. 3. — Metastatic anaplastic carcinoma in liver.

tissue deposits, varying in size from 1 to 10 mm. in diameter. The tumor was elevated above the cut surface and was firm, granular, and reddish-gray. The hilar lymph nodes were considerably enlarged and on section appeared to be completely replaced by tumor tissue. The largest lymph node measured 4 cm. in diameter. The liver weighed 2,000 gm. The surface was dark reddish-brown, and numerous small umbilicated tumor masses were visualized. On section, the liver showed small and large tumor masses within the deeper portion of the organ. The largest tumor deposit was 3 cm. in diameter. There was some necrosis in the central portion of these areas. There were some small tumor deposits on the surface of the left ovary. On histological examination of the excised structures, there were found to be microscopic foci of tumor in the cervical esophagus, bronchi, bone, and muscles. Section of the tumor in the larynx showed the neoplasm, which appeared to arise in the mucous membrane and extend through the entire thickness of the wall. The tumor consists of spindle and oval-shaped cells which were relatively small and showed almost complete filling of the cell by the large nucleus. The nuclei were dark-staining, and many mitotic figures were present. Histologic examination of the lungs, liver, lymph nodes, bone, muscle, bronchi, esophagus, and ovary showed the invasion by the same type of tumor (Fig. 3). The final anatomical diagnosis was small-cell carcinoma of the larynx with extensive metastases.

Comment

In view of the comparative rarity of tumors of the larynx in women (particularly in women of this patient's age) and the unusually high grade of malignancy of this tumor, it is natural to speculate as to its possible origin. The recent pregnancy may, in some way, be related to the more malignant character of the tumor. One might speculate concerning the relationship of the previous x-ray therapy as an etiological factor in the development of this tumor. Recently, many reports of tumors of skin and also of deep structures are noted after therapeutic radiation for other purposes. Although cutaneous carcinomas are the most frequent, many deep lesions have been recorded. It does seem, however, that squamous epithelium is more susceptible to radiation malignancy than other forms of tissue.

Unfortunately, we have been unable to ascertain the total roentgen dosage received by this patient. However, according to the history, it was very extensive. Radiotherapy to the thyroid gland for goiter has been used in the past, although at present it is not considered good treatment. It is possible that some of these people will develop neoplastic lesions in the cervical tissues, such as the larynx, as a result of this therapy. This case was further complicated by the known history of tuberculosis, which tended to form the opinion that the laryngeal lesion was also due to this cause.

Summary

A case history of a highly malignant spindle-cell type of carcinoma of the larynx in a relatively young, white woman is recorded.

The rarity of this type of neoplasm is established.

Conjecture is made concerning the relationship of this tumor to previous x-ray therapy.

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Tumors of the Temporal Bone

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We have classified under the heading of tumors of the temporal bone all neoplasms known in the past as "tumors of the middle ear," "tumors of the middle and external ear," "tumors of the petrous bone," and "tumors of the mastoid process."

All pathologic processes affecting the temporal bone, because of the variety of anatomical formations of the region, give rise to a group of clinical and pathological signs which may be considered as characteristic and constant.

Primary and secondary tumors are differentiated as follows: All new growths affecting, exclusively or preeminently, the temporal bone structure or the surrounding vessels, nerves, and muscles, as well as the ones penetrating into the temporal bone, are considered primary. Therefore, the primary tumors are epithelioma and connective tissue tumors of the middle ear, true cholesteatoma, tympanic and jugular paraganglioma, neurinoma of the facial and acoustic nerve, and meningiomas.

Secondary tumors are tumors of the external ear, of the skin of the periaural region, of the parotid gland or the nasopharynx, or intracranial tumors, such as neurinoma of the acoustic nerve, meningioma, and some types of jugular paraganglioma that have their original sites in anatomical formations not related to the temporal bone and that affect the latter only in part, during the later stages of their growth or by a metastatic process.

Pathogenesis and Topographic Aspects

The temporal bone is formed by an external part and an internal part. In the external part is found the region of the tympanic cavity, the mastoid, and the squamous portion of the temporal bone. In the internal part is found the petrous bone.

Benign tumors usually remain localized in one of these parts, while the malignant

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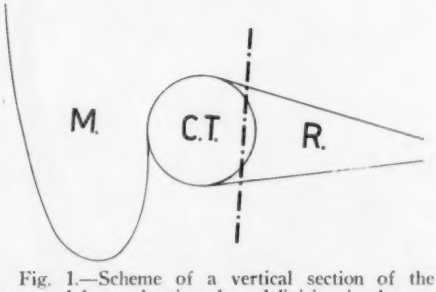


Fig. 1.—Scheme of a vertical section of the temporal bone, showing the subdivision in the external and the internal part. The dotted line shows the partition between the external and the internal part. *M.*, indicates mastoid; *C. T.*, tympanic cavity; *R.*, petrous bone.

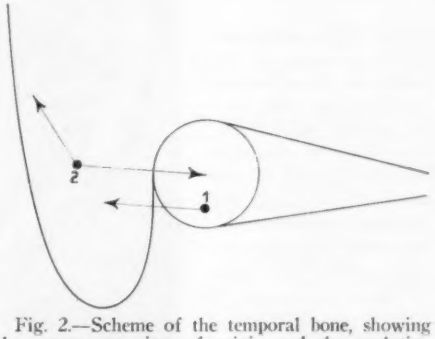


Fig. 2.—Scheme of the temporal bone, showing the commonest sites of origin and the evolution of benign epithelial and connective tissue tumors of the middle ear. The evolution is constantly an external one.

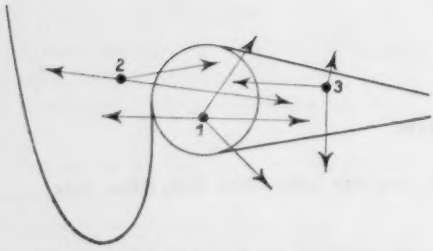


Fig. 3.—Scheme of the temporal bone, showing the commonest points of origin and the evolution of malignant epithelial and connective tissue tumors of the middle ear. Both the internal and the external parts are attained.

growths and paraganglioma show a tendency to spread.

It has been shown that malignant growths arising in the external part spread to the internal part, while benign tumors remain localized to the external zone. All tumors of the internal part show a tendency to grow in a lateral direction, affecting the external portion of the temporal bone. Paraganglioma, having an intermediate origin between the two parts, spreads at the same time in both directions.

Based on operative or autopsy findings, or on otoscopic, radiologic, and neurologic signs, every tumor of the temporal bone, including those secondary forms, may be classified in one of these groups.

Not all the cases reported in the literature, however, can be grouped in this way, as the radiologic investigation of most of them, especially the internal forms, are not adequate.

From a practical point of view, it is evident that an efficient surgical therapy is possible only for tumors of the external part of the temporal bone.

Clinical Aspects

The following clinical aspects of tumors of the temporal bone have been considered: otological, radiological, and neurological.

These aspects may be very easily studied in tympanojugular paragangliomas. These tumors, well localized from their onset, show a very slow growth process. This allows for a differentiation between various types of symptomatology. Therefore a radio-otoscopic syndrome (Chaussé, 1947), an external and an internal variety (Cova and Meda, 1951; Cova, Meda, and Nicelli, 1952), a radio-otoscopic syndrome of the tympanum (Chaussé, et al. 1953), as well as one of the jugular foramen and a jugular-tympanic syndrome, have been described. Bickerstaff and Howell, in 1953, reported on four types according to the succession of symptoms and the site of origin.

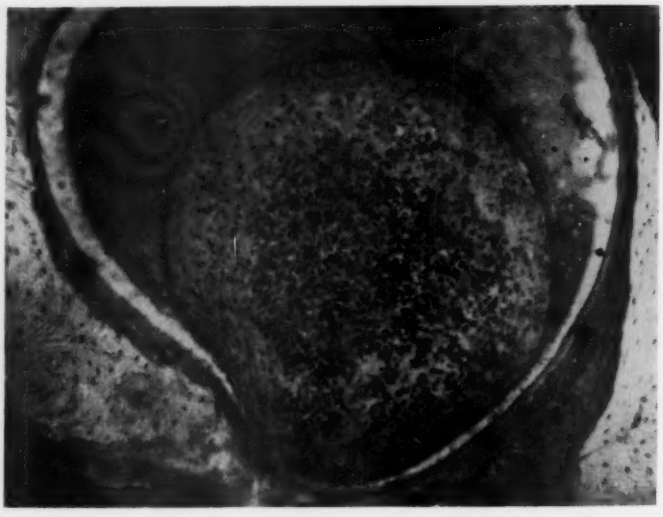


Fig. 4.—Polymorphous-celled sarcoma of the middle ear. Round-shaped polymorphous cells form the growth which originates from the submucosa and is covered by the epithelial layer of the mucous membrane. Hematoxylin and eosin; reduced 10% from mag. × 80.

TABLE 1.—*Clinical Syndromes in Tumors of the Temporal Bone*

Syndrome	Lesion		
	Tympanic	Radiological	Neurological
Otoradiological	+	+	−
Otoneuroradiological	+	+	+
Otoneurological	+	−	+
Neuroradiological	−	+	+
Otological	+	−	−
Neurological	−	−	+
Radiological	−	+	−

It should be pointed out once more that no attempt at classification of these tumors is possible without the aid of tomography and the special projections for the jugular foramen, which are of great help—particularly in the cases of tumors of the internal part.

The different syndromes presented in these patients and their order of succession are shown in Table 1.

It should be pointed out that these associations of symptoms vary according to the pathology of the tumors. In cases of benign growths, the modifications are slow and gradual. In case of malignant tumors, there are sudden changes in the clinical picture.

When the tympanic picture does not offer any clear-cut orientation of the tumor pathology, we may, guided by the succession of symptoms, venture an opinion on the extension, localization, and pathology of the tumor. The scheme we propose is best for the initial stage of the tumor growth when the otological, neurological, or radiological syndromes alone are present.

Classification

In Table 2 is shown the histological classification that has been adopted for tumors of the temporal bone. Figure 2 is a schematic drawing of the most common sites of origin of the primary tumors of the temporal bone.

Radiological Aspects

The semeiologic possibilities of radiology have improved in the last few years, thanks

Fig. 5.—Reticulum-cell sarcoma of the right temporal bone. Paralysis of the right hypoglossal nerve. Lower right, the histological aspect of the tumor. Hematoxylin and eosin; reduced 25% from mag. × 75.

to the use of tomography, to the possibility of enlarging x-ray pictures, and to the use of x-ray tubes having a moving anode with a very small target. In addition, stereoradiography and the use of some of the new radiographic projections permit the recognition of nearly all the details of the temporal bone.

For these reasons, a radiographic examination, especially a tomographic one, is now

TABLE 2.—*Histological Classification*

Epithelial tumors	Benign forms & types showing local malignancy
True cholesteatoma	Malignant forms
Connective tissue tumors	Benign forms & types showing only local malignancy; malignant forms
Bone tissue tumors	Benign forms & types showing only local malignancy
Nervous tissue tumors	Paraganglioma; neurinoma; meningioma; ependymoma
Metastatic tumors	
Systemic blood diseases	

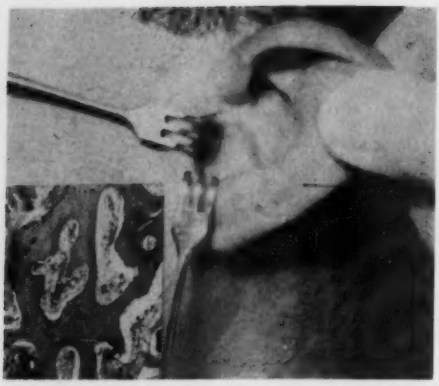


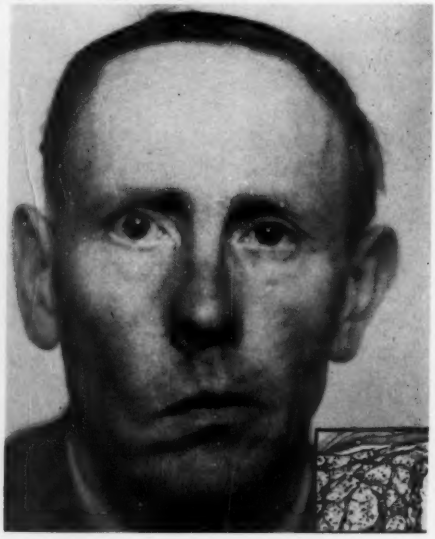
Fig. 6.—Spongy osteoma of the left external auditory meatus. Lower left, the microscopic picture; reduced about 10% from mag. × 20.

necessary, not only in the presence of gross lesions but also in the early stages of the tumor growth. In the latter case, tomography is a great help in evaluating the site of the lesion and its extension. This allows an interpretation of other otological symptoms and the completion of the clinical picture. Chaussé's second projection for the jugular fossa should always be employed, as it may add some details which cannot be shown even by tomography.

Fig. 8.—Right tympanojugular paraganglioma; paresis of the 7th and 11th nerves (atrophy of sternocleidomastoid and trapezius muscles) and of the 10th nerve (laryngeal hemiplegia).

Fig. 7.—Right tympanic paraganglioma; otoscopic view of the new growth.

Fig. 9.—Tympanojugular paraganglioma; paralysis of the seventh nerve, which persists after clinical healing following roentgen therapy. Lower right, histological aspect.



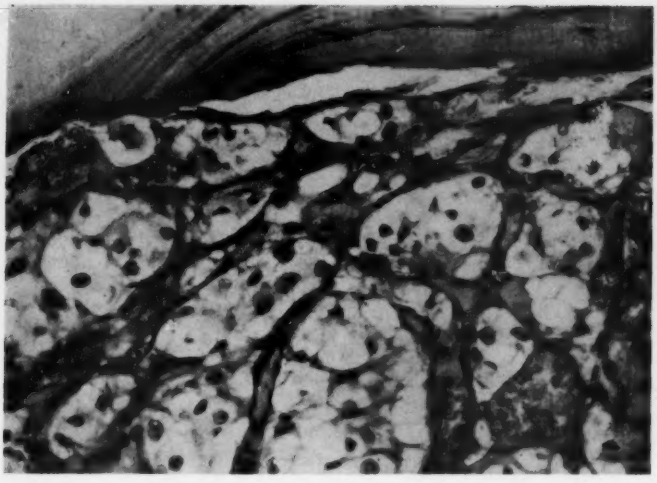


Fig. 10.—Tympanojugular paraganglioma; typical microscopical aspect of the tumor affecting the temporal bone.

The x-ray picture of the temporal bone which is being invaded by a new growth is always the same. In the case of primary and secondary tumors, there is a destruction of the bone due to the osteolytic activity of the tumor. It should be remembered, however, that metastases from tumors of the prostate, the thyroid, and the breast are usually of the osteoplastic type. The granular appearance of bone structures, which is often observed in tumors of the temporal bone, may be sometimes related to a true neoplastic infiltration, whereas in other cases it is conditioned by a concomitant inflammatory process.

There is no possibility of differentiating chronic destructive inflammatory forms from initial neoplastic processes. The latter often show a contemporary presence of inflammation. Extensive lesions where the surrounding bone tissue shows an erosive aspect are usually due to neoplastic process.

Epithelial Tumors

Benign Tumors and So-Called Forms with Local Malignancy, Though Histologically Benign.—In this group are papillomata, mixed tumors of the seromucous glands, and cylindromata. These are all extremely rare tumors, even rarer than benign connective tissue growths.

Owing to the external spreading of these growths, the clinical picture presented is nearly always that of a pure otological syndrome or an otoradiological syndrome.

Radical surgery should be adopted as the treatment of these forms.

Carcinoma.—These growths originate from the epithelium of the tympanic cavity or the mucosa of the paratympanic cavities. It is agreed that a chronic discharge from the ear might favor, through its irritative action, the onset of a malignant growth. In nearly all cases these tumors are squamous carcinomata, but the occurrence of basal-cell carcinoma and transitional-cell epithelioma has been reported.

The evolution of the tumoral growth does not follow any fixed direction. The neo-

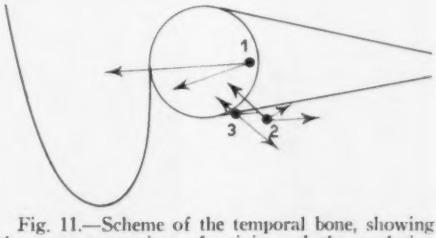


Fig. 11.—Scheme of the temporal bone, showing the commonest sites of origin and the evolution of tympanojugular paragangliomas. Forms originating from the tympanic paraganglia have an external evolution (*1*); those originating from the jugular paraganglia (*2*) and the tympanic canal (*3*) have both an internal and an external evolution.

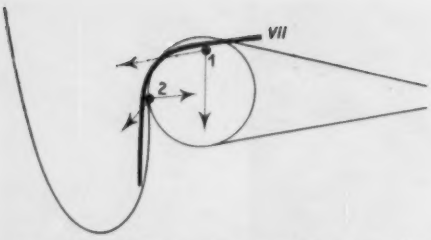


Fig. 12.—Scheme of the temporal bone showing the sites of origin and the evolution of neurinoma of the facial nerve (VII). The evolution is always an external one.

plasms invade the aural cavities and then, eroding the surrounding bone, reach the mastoid, the jugular foramen, and the middle and posterior cranial fossae.

The first period of growth of these tumors may be characterized by otoscopical, otoradiological, or otoneurological signs. In a later stage of evolution, the external or internal, or even both, types of the otoneuroradiological syndrome may be observed.

The two surgical procedures employed nowadays for these tumors are (1) local surgery with the use of the technique of a common radical mastoid with demolition depending on the extension of the growth of the whole mastoid and the petrous bone, followed by electrocoagulation of the operation cavity, and (2) en bloc removal of the greatest possible part of the temporal bone, comprising the region of the middle ear, where the neoplasm has arisen.

When the external ear has become affected primarily or secondarily, the whole of the external auditory meatus, alone or with the pinna, must be taken away. The same principle applies to the other surrounding structures, such as the parotid gland, the condyloid process, and the zygomatic arch. The involvement of the cervical lymph nodes does not occur often enough to justify a prophylactic block neck dissection. However, on this point there are divergent views, and the problem cannot be considered settled.

True Cholesteatoma.—This tumor, which has been called "pearl tumor," "primitive cranial epidermoid," "keratoma," "epidermoid cyst," and "inclusion cyst," is a very uncommon growth.

A true cholesteatoma is a growth of the temporal bone due to a dysontogenetic process, owing to the inclusion in the bone structures of ectodermic elements derived from the first branchial arch. This interpretation draws further support from reports of association of true cholesteatoma with other malformations of the middle and internal ear and of the temporal bone. The other malformations have been dysplasia auris, dysontogenetic cysts, fistulas, and coloboma. Therefore, true cholesteatoma

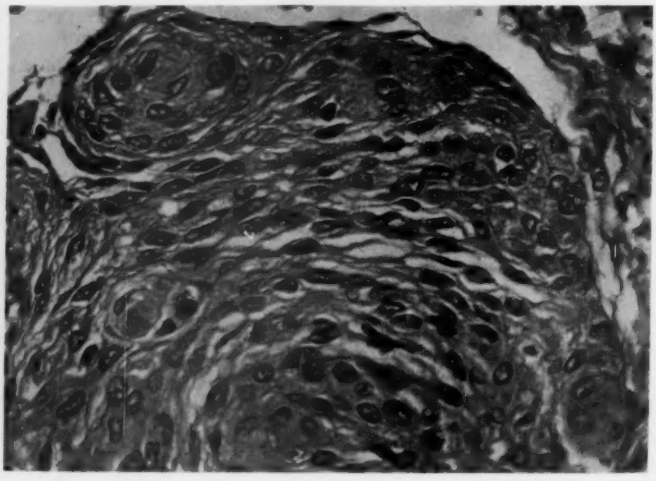


Fig. 13.—Meningioma of the temporal bone; the cells show a vortex-like distribution. The cytoplasm is partly occupied by fibrils.

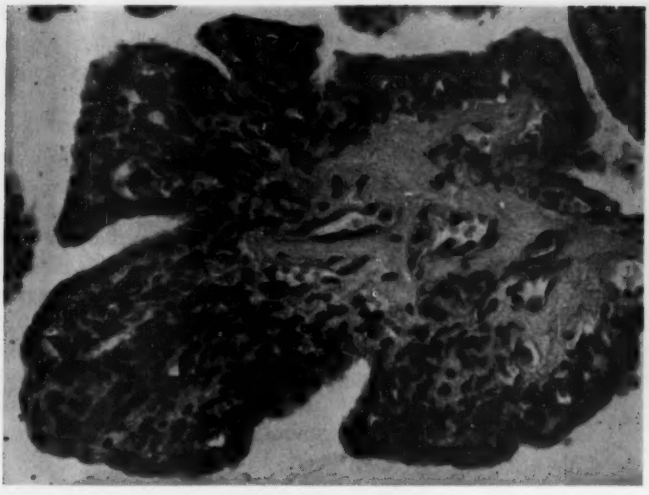


Fig. 14.—Ependymoma of the temporal bone. Note the large amount of blood vessels and the cylindrical-cubic covering.

should be considered as a benign dysontogenetic new growth.

The symptoms are characterized by neurological, otoradiological signs and often by external otoneurological signs. Intracranial forms may show a radiological or an internal neuroradiological group of symptoms.

Connective Tissue Tumors

Benign Tumors and So-Called Forms with Local Malignancy, Though Histologically Benign.—Representatives of the first group are fibroma, myxoma, chondroma, and angioma; for the second group, fibrosarcomas and myoblastomyomas.

These tumors usually affect the external part and give a pure otoscopic or, in some cases, an otoradiological syndrome.

Radical surgery is the only type of treatment for these benign tumors.

Malignant Connective Tissue Tumors.—Round-cell tumors of the immature type are the common varieties of sarcoma of the middle ear. It is commonly believed that some of these tumors may derive from remnants of the mucous mesenchymal tissue which occupies the middle ear of the newborn and persists there for some months after birth. Retothelial sarcoma is considered exceptional.

Owing to the fact that these tumors show an extremely rapid evolution and spread in all directions, pure otoscopic or otoradiological signs are seldom found. Instead, an external-internal otoneuroradiological syndrome may be generally observed.

As these growths show a marked radiosensitivity, they should always be treated by irradiation.

Fig. 15.—Jugular paraganglioma. Hirtz's axial projection of the skull. The left petrous bone is much thinner, owing to the erosion which affects further the walls of the anterior and posterior lacerated foramen, which are thus larger than usual. The destructive process affects in part the occipital condyle. Note further that the foramen of the vertebral artery at the level of the left lateral mass of the atlas is very large.

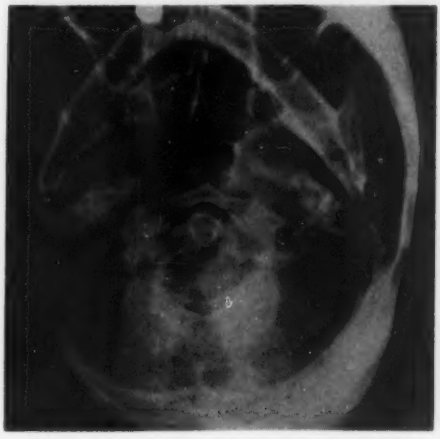


Fig. 16.—Jugular paraganglioma. Horizontal tomograms at the level of the posterior lacerated foramen (Hirtz's projection). The design of the posterior lacerated foramen appears to be clearer than in the second projection of Chaussé. The said foramen is much enlarged, owing to the regular erosion of its contours. No invasion of the bone, as can usually be seen in malignant tumors. The largest area of destruction affects the apex of the petrous bone and the occipital condyle, which has nearly disappeared. The partition between the posterior lacerated foramen and the occipital foramen is very thin.

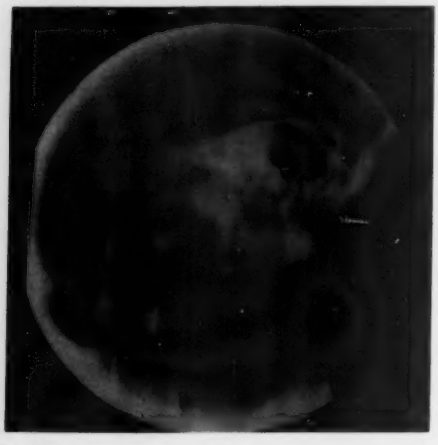
Bone Tissue Tumors

All varieties of bone tumors can be observed: benign osteoma, myeloplax tumors showing a low degree of malignancy, and highly malignant Ewing's sarcomata.

Osteoma is rather frequent, for included in this group are those forms which had formerly been classified as exostoses. The other varieties mentioned above constitute exceptional findings.

These tumors give rise to pure radiological or to otological syndromes or to a mixed otoradiological syndrome. Malignant forms show otoradiological or otoneuroradiological symptoms. Surgical therapy gives good results in benign tumors. Surgery, alone or combined with radiosurgical treatment, or irradiation alone have been employed for malignant tumors, but the results have been discouraging.

Fig. 17.—Jugular paraganglioma. Frontal tomogram, showing extensive erosion of the petrous bone, which appears to be shortened. The medial part of the jugular fossa is enlarged, whereas its lateral aspects retain their normal shape. The tympanic cavity is not affected.



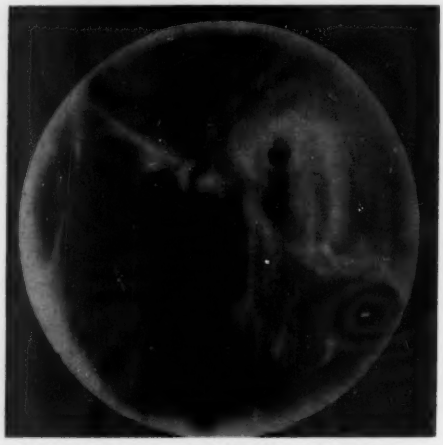


Fig. 18.—Jugular paraganglioma. Sagittal tomograms show that the petrous bone is somewhat suspended, owing to erosion of the peripheral structures of the jugular fossa, which appears to be considerably enlarged. The erosion has caused a partial destruction of the bone capsule of the basal turn of the cochlea and, partially, of the inferior half of the posterior semicircular canal.

Nerve Tissue Tumors

This group comprises tympanojugular paraganglioma, neurinoma, meningioma, and ependymoma.

Tympanojugular Paraganglioma.—This type of new growth was observed by Lubbers, in 1937, and later, in 1945, by Rosenwasser, who called it "tumor of the ear and mastoid of carotid body type." It is also

known under the names of "glomus jugulare tumor" (Winship, Klopp, and Jenkins, 1948, Magarey, 1952, Terracol and Guerrier, 1956), "nonchromaffin paraganglioma" (Lattes and Waltner, 1949; Dockerty, Love, and Patton, 1951), "tympanic body tumors" (Lundgren, 1949; Berg, 1950), "glomerocytoma tympanicum" (Zettergren and Lindström, 1951), "tympanic-jugular glomic tumor" (Cova and Meda, 1951; Cova, Meda, and Nicelli, 1952), "jugular receptoma" (Gaffney, 1953), "glomus jugulare tumor" and "chemodectoma of the glomus jugulare" (Figi and Weisman, 1954; Huppler, McBean, and Parkhill, 1955; Williams, et al., 1955), "tympanojugular paraganglioma" (Meda, Leonardelli, and Cova, 1955), and tumors of the "vasoregulator tissues" (Sirtori and Leonardelli, 1958).

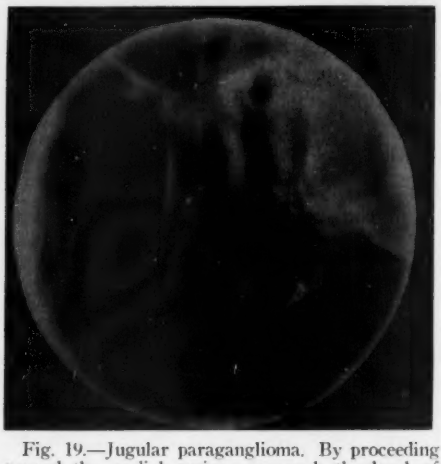


Fig. 19.—Jugular paraganglioma. By proceeding toward the medial region, we reach the level of the vestibulum. The marked enlargement of the jugular fossa is even more evident.

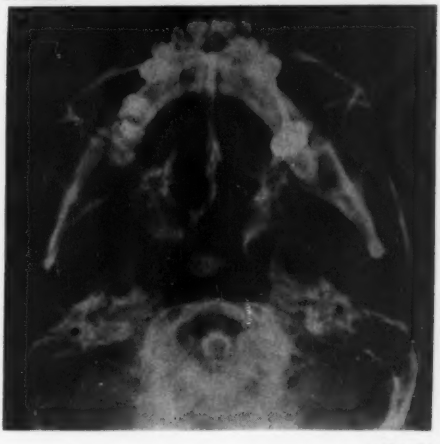
By critically reviewing the literature it was noted that these tumors have frequently been mistaken for other neoplasms (adenoma, fibrous angioma, angioendothelioma, meningioma, etc.) and for simple hyperplastic inflammatory changes in the tissues of the region.

As far as the pathogenesis of these tumors is concerned, it is now agreed that they are derived from the tympanic and jugular

Fig. 20.—Jugular paraganglioma. Second projection of Chaussé. The anterior lacerated foramen is much enlarged; the partition with the occipital foramen is very thin. There is an erosion of the occipital condyle at the level of the outlet of the hypoglossus canal.

paraganglia situated along the external layer of the superior bulb of the internal jugular vein near the nerve endings of Jackson's plexus. These paraganglia can be considered

Fig. 21.—Tympanojugular paraganglioma. Axial view of the skull (Hirtz's incidence). The bony structure of the floor of the right middle cranial fossa has nearly disappeared. Similarly, there are few traces left of the walls of the bony portion of the tube. Conversely, the tympanic bone has a spotted, condensed aspect, showing a condition of damage. The pyramid of the anterior profile of the temporal bone is partly destroyed. Near the apex there exists a large incision reaching laterally the capsule of the medial turn of the cochlea.



to be part of the group of organs having a neurovascular structure, which exert a chemopressor regulating activity.

From a histological point of view, these tumors have often been considered benign. However, because of the damages produced by their spreading to the intracranial structures, they have also been considered malignant. Reports of metastases to the cervical glands (Winship, Klopp, and Jenkins, 1948), to the liver (Lattes and Waltner, 1949), to the lung (Tamari, McMahon, and Bergendahl, 1951), and to the oral cavity (Bradley and Maxwell, 1954) have lead to the belief that the tumors are potentially malignant. These growths usually spread along the lines of least resistance: to the tympanic cavity and the paratympanic region when they originate from the tympanic paraganglia (tympanic paraganglioma), to the jugular foramen (jugular paraganglioma), and to both regions when the new growths arise from the paraganglia of the carotid-tympanic canal (tympanojugular paraganglioma).

The symptoms, which vary according to the site of origin and to the topographic evolution, are shown in Table 3. The classifications of Cova, Meda, and Nicelli and the classification of Bickerstaff and Howell distinguish three types of tympanojugular paragangliomas: "external" or tympanic ones, "internal" or jugular ones, and "mixed" or external-internal-tympanojugular types.

From the point of view of therapy, surgical treatment gives the best results for these tumors. However, there are great difficulties, owing to the profuse hemorrhages which arise during surgical intervention. In external tumors affecting only the middle ear, radical surgery followed by coagulation and then by radiotherapy can be considered the best form of therapy. Local recurrences in the operation cavity may be treated in the same way (Pietrantoni, 1948). Internal and mixed forms cannot be operated on because the region of the jugular foramen cannot be reached without endangering vital anatomical structures (Bickerstaff and Howell, 1953; and Capps, 1953). However, good results have been reported with x-ray therapy.

TABLE 3.—*Symptomatology of Tympanojugular Paragangliomas*

External Evolution	Internal Evolution
Tinnitus	Paralysis of nerves of base of skull
Deafness	Intracranial or cervical evolution
Vertigo	Internal radiological lesion
Tympanic hyperemia	
Facial paralysis	
Discharge of growth in external meatus or behind ear	
External radiological lesion	

Fig. 22.—Tympanojugular paraganglioma. Oblique view of the skull (Schüller's view). The squama of the temporal bone in front of the pyramid on top of the zygomatic arch shows widespread irregular osteolysis processes. Some normal bone areas can still be seen. The borders of the osteolytic zone are clear-cut. There exists a rarefaction process of the posterior pillar of the zygomatic arch.

Neurinoma. — Neurinomas originating from the facial and from the acoustic nerve as well, and having the same histological characteristics, have been described, but they occur very rarely.

Neurinoma of the facial nerve give constant and long-lasting symptoms. The commonest signs of the presence of a neurinoma are complete paralysis and nervous dyskinesia, which may be of sudden or gradual onset and affect the mimic muscles of the face. Neurinoma of the horizontal portion of the facial nerve, reaching the chorda tympani, may be accompanied by an altera-

tion of taste of the corresponding part of the tongue.

Neurinomas of the acoustic nerve affect the posterior cerebral fossa and are accompanied by neuroradiological, neurological, or purely radiological signs of the internal type. Both neurinoma of the facial and of the acoustic nerve should receive surgical treatment.

Meningioma.—Only a few cases of meningioma spreading to the temporal bone have been described. They show a slow progression and, if the middle ear is attained, the clinical picture of chronic otitis is the commonest symptom. Thus may be seen an otoradiological syndrome, a pure radiological one, or an otoneuroradiological one. Surgery appears to be the best treatment of these forms.

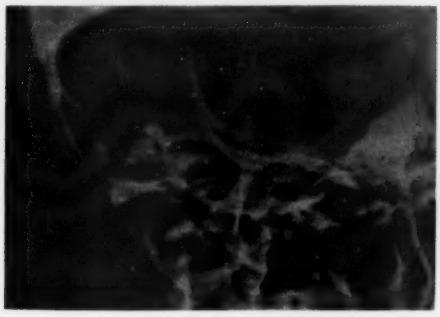


Fig. 23.—Tympanojugular paraganglioma (Stenver's projection). This view does not allow a minute investigation. Only spotted areas can be seen in the anterior and posterior bone walls of the tympanic cavity. Further, there are signs of partial destruction of the inferior bone capsule of the basal turn of the cochlea. No good view of the attic can be obtained. The niche of the round window appears to be enlarged.

Ependymoma.—This tumor, which has not been observed in the temporal bone by others, was studied by us in one case. This case showed an external and internal neuroradiological syndrome without any sign of tympanic lesions.

Metastatic Tumors.—Metastases of other tumors in the temporal bone have been described in only a few cases. They show a silent evolution (Altmann, 1931). A search of the literature showed reports of metastases in the temporal bone, originating from

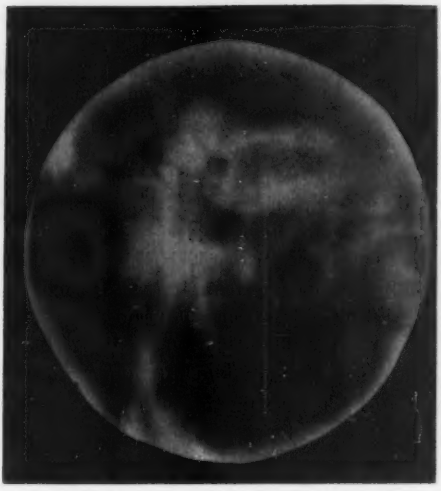


Fig. 24.—Tympanojugular paraganglioma. Frontal tomogram of the anterior part of the petrous bone; destruction of the bone partition between hypotympanum, jugular fossa, and carotid canal. The bony Eustachian tube appears to be nearly completely destroyed.

the following primary tumors: hypernephroid tumors, breast adenoma and adenocarcinoma, prostatic carcinoma, adenocarcinoma of the stomach and of the thyroid, laryngeal carcinoma, seminoma, lymphosarcoma of the intestine.

Fig. 25.—Tympanojugular paraganglioma. The sagittal tomograms show an erosion of the anterior wall of the petrous bone. Beside the profile the floor of the middle cranial fossa is less opaque.

TABLE 4.—*Case Material*

Case	Sex	Age	Histology	Clinical Signs	Type of Syndrome	Results
1	M	50	Spinocellular carcinoma	Chr. ear discharge; granulations, paresis of VII, VIII, X, XI	Ext. & int. otoneuroradiological	Dead
2	F	53	Spinocellular carcinoma	Sequelae of rad. mastoid op.; granulations; paresis of I, V, VI, VII	Ext. & int. otoneuroradiological	Dead
3	F	30	Spinocellular carcinoma	Purulent-bloody discharge for years; fistula of mastoid	Ext. & int. otoradiological	Dead
4	F	80	Poorly differentiated carcinoma	Polyps with purulent & hematic discharge; paresis of VI	Ext. otoneuroradiological	Unknown
5	F	51	Spinocellular carcinoma	Ear discharge for 2 yr.: sequelae of recent rad. mastoid; granulations	Ext. & int. otoradiological	Unknown
6	F	42	Poorly differentiated carcinoma	Chr. discharge; granulations; pharyngeal abscess, paresis of VII	Ext. & int. otoneuroradiological	Dead
7	M	60	Spinocellular carcinoma	Chr. discharge; petrositis; cerebral abscess; paresis of VII	Ext. & int. otoneuroradiological	Dead
8	F	4	Polymorphous sarcoma	Polyps for months; paresis of VII & VIII, cerebellar syndr.	Ext. & int. otoneurological	Dead
9	F	66	Reticular sarcoma	Polyps for years; paresis of XII	Ext. & int. otoneuroradiological	Alive after 5 yr.
10	M	48	Osteoma	Stenosis of ext. meatus.; chr. discharge	Ext. otoradiological	Alive after 3 yr.
11	F	60	Osteoma	Stenosis of ext. meatus	Ext. otoradiological	Alive after 3 yr.
12	M	24	Osteoma	Stenosis of ext. meatus	Ext. otoradiological	Alive after 3 yr.
13	F	47	Paraganglioma	Polyps for years; paresis of VII	Ext. otoneuroradiological	Alive after 14 yr.
14	F	53	Paraganglioma	Polyps; discharge for years; paresis of VII; paralysis of VIII, X, XI, XII	Ext. & int. otoneuroradiological	Alive after 6 yr.
15	F	27	Paraganglioma	Polyps; chr. discharge; paresis of VII; paralysis of XII	Ext. & int. otoneuroradiological	Alive after 6 yr.
16	F	52	Paraganglioma	Polyps; chr. discharge; fistula under ear; paresis of VII	Ext. otoneuroradiological	Alive after 6 yr.
17	M	40	Paraganglioma	Polyps; chr. discharge; paralysis of VII:	Ext. & int. otoneuroradiological	Alive after 8 yr.
18	F	34	Paraganglioma	Polyps; paresis of VII	Ext. otoneurological	Alive after 2 yr.
19	F	45	Paraganglioma	Swelling & cong. of lower tympanic region; paresis of VII	Otological - later ext. otoneurological (?)	Alive after 6 yr.
20	M	39	Paraganglioma	Polyps for years; paralysis of VII	Ext. otoneuroradiological	Alive after 2 yr.
21	F	27	Paraganglioma	Swelling & cong. of lower tympanic region	Ext. & int. otoradiological	Alive after 3 yr.
22	F	65	Paraganglioma	Polyps; chr. discharge; paralysis of VII	Ext. (?) otoneurological	Alive after 4 yr.
23	M	54	Paraganglioma	Inf. tympanic polyps; purulent discharge	Ext. & int. otoradiological	Alive after 4 yr.
24	F	64	Paraganglioma	Polyps; purulent discharge	Ext. & int. otoradiological	Alive after 3 yr.
25	M	35	Paraganglioma	Chr. discharge, paralysis of VII	Ext. & int. otoneuroradiological	Alive after 3 yr.
26	F	55	Paraganglioma	Polyps; hematic discharge spreading to post. cran. fossa; paralysis of VII, XII	Ext. & int. otoneuroradiological	Alive after 6 mo.
27	F	45	Jug. paraganglioma	Pharyngeal swelling; paralysis of X, XI	Int. neuroradiological	Alive after 2 yr.
28	M	70	Paraganglioma	Parotid swelling; paralysis of VII	Ext. & int. otoneuroradiological	Alive after 6 mo.
29	F	49	Neurilemmoma of facial nerve	New growth of ext. meatus; chr. discharge; paralysis of VII	Ext. otoneuroradiological	Alive after 6 mo.
30	F	44	Meningioma	Purulent discharge; granulations	Ext. & int. otoradiological	Alive after 3 yr.
31	M	32	Ependymoma	Paralysis of VII	Ext. and int. neuroradiological	Unknown
32	F	53	Metastasis from breast carcinoma	Purulent discharge; metastatic spread to bones	Ext. & int. otoradiological	Unknown

Further, it is known that leukemia and systemic lymphosarcoma may infiltrate the temporal bone.

Summary

Under the heading of "tumors of the temporal bone," a classification is given of all neoplasms which originate from the structures of the temporal bone and from those closely related to it.

The temporal bone may be subdivided into an external part, constituted by middle ear and mastoid process, and an internal one, constituted by the petrous bone. All these tumors show a starting point and an evolution, which are constantly related to these anatomical parts. This fact justifies the grouping in internal and external forms.

From a clinical standpoint, three groups of symptoms may be taken into account: otoscopic, neurologic, and radiologic signs. The different association of these symptoms gives rise to corresponding different syndromes.

Personal cases are listed which illustrate the various tumor types developing in this site and their pathological, clinical, and therapeutical characteristics.

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Binaural Hearing and the Hearing-Impaired

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I

Within the past few years, there has been a renewed and vigorous attempt to reproduce by unique electromechanical methods our multidimensional acoustic environment. On one hand, there has been the influx of stereophonic sound in the attempt to reproduce the "sound space" attendant to original listening conditions. A second interest, implemented by electronic miniaturization, is the binaural hearing aid. Each of these approaches reflects an attempt to provide both the normal and the hearing-impaired listener with a more complete auditory experience than previously was possible with a single reproduction or amplification system. The purpose of this present discussion is to call attention to what appears to be many neglected aspects and pitfalls in applying binaural amplification to the hearing-impaired and to scrutinize, in light of previous experimentation, the current clinical methods frequently used in the evaluation and selection of hearing aids in general and binaural hearing aids in particular.

II

One source of confusion in understanding binaural listening, especially among the hearing-impaired, seems to be a lack of awareness of the similarities and differences among (1) stereophonic *sound,* (2) binaural *sound,* and (3) binaural *listening.* The following discussion will attempt to show that there is no such thing as stereophonic *listening.* A clear delineation is needed between what is being responded to and the requisites necessary for the observed responses.

Very often, both stereophonic and binaural sound are considered to be identical when, in fact, they represent two entirely separate concepts. The apparent similarity between these two systems of sound reproduction is their ability to create the illusion of a more adequate representation of an original acoustic event than is possible with the conventional single-channel system. The differences between stereophonic and binaural sound are reflected by their different methods of recording and reproduction.

The aim of stereophonic sound is to create for the listener a sound space which is a faithful re-creation of an original acoustic event. This restoration may be accomplished in many different ways. In the main, two independent recording and reproduction systems are used. The stereophonic re-creation of a sound space then may be achieved by playing the independent recordings through two independent loud-speakers. At first glance, this may appear to be straightforward and involve little more than a dual-channel system. Closer inspection, however, reveals some subtle considerations. One is the placement of the loud-speakers in relation to the physical characteristics of the room. The speakers must be sufficiently spaced to create the desired illusion but not so distant that one sound or set of sounds can be associated with only one of them.

A second consideration is the method used to record the original acoustic event. More specifically, the quality of a three-dimensional space is dependent upon the distance

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between the microphones used in the original recording. In many ways, proper control of microphone placement has become the "art" of stereophonic recording.

A basic difference between stereophonic and binaural sound is the placement of the recording microphones. In binaural sound, the recording microphones are set approximately 8 in. apart to reproduce the distance between the ears of the average listener. Reproduction of binaural recordings then may be effected through earphones. When the binaural recording is played back through two independent loud-speakers, the illusion of a sound space still may be achieved but only within a limited area between each speaker. When the listener moves outside the circumscribed binaural-listening area, the three-dimensional effect is lost and one loud-speaker predominates.

The normal-hearing listener, or receiver of these complex patterns of auditory stimuli, is binaural in character. He is a binaural listener whether the complex auditory stimuli originates by binaural or stereophonic reproduction methods or occur in his everyday environment. To restate, the normal listener is a binaural receiver of sound coming from one or more sources. It just happens that it is possible to create the illusion of a sound space by binaural or stereophonic recording and reproduction techniques. Furthermore, within the context just described, use of the phrase "stereophonic listening" only tends to confuse an already sufficiently complex subject.

Briefly, binaural and stereophonic sound have some similar and dissimilar properties. It is important to be aware of these factors and their relation to binaural listening in order to maintain each in its proper perspective. Considerable confusion can result in blithely reasoning from one to the other or by using terminology implying one but meaning the other.

III

In binaural listening, the discrimination, identification, and the subsequent localization or selection of a particular sound source within an acoustic environment containing many different auditory stimuli is not an extremely difficult task for the normal listener in most of his everyday listening situations. Three major parameters of the auditory stimulus that act as clues for the normal listener in his localization of a particular sound in space are intensity, time (phase), and spectral composition.

When the intensity of a sound is greater at one ear, the sound appears to be on that side where it is presumably also louder. Similarly, the direction of a sound appears to be on the side that first receives the stimulus. In general, intensity appears to be the predominant factor for the higher frequencies (above 1,400-1,500 cps) and time the predominant factor for the lower frequencies (below 1,400-1,500 cps).[23,27,31-33] On the other hand, it is interesting to note that when a stimulus contains both high- and low-frequency elements, such as clicks or broad-band noise, it is possible to use intensity to compensate for time and time for intensity in maintaining the same apparent location of an acoustically complex signal.[32]

This duality of function of intensity and time seems to illustrate, as a first approximation, the complexity of binaural listening. The additional factor, still a first approximation, is the spectral composition of the sounds reaching each ear. Interposition of the head between the ears in a sound field creates a difference at each ear in the relation between the individual sounds constituting a complex auditory stimulus, like speech.[15,28,32,35] As a consequence, the quality of the sound at each ear will appear to be different. These quality differences may not be immediately apparent to the normal binaural listener in a sound field, but with only one useful ear it is frequently possible to localize the sound source.[1,12,22,24]

To recapitulate, the listener with normal hearing in each ear in a sound field is a binaural receiver. As such, he should be

able to take advantage of the minimal differences in sound patterns impinging on each of his ears. In addition, it is assumed that he is a sufficiently intact organism to be able to interpret these different sound patterns and thereby localize, as well as differentiate, complex auditory stimuli. When the total complex of binaural listening is considered, the vestibular and proprioceptive cues associated with head movement must not be neglected as contributing factors.

The listener with a unilateral or bilateral hearing loss is not a binaural receiver in the same way as are normal hearing persons. Persons with only one ear severely impaired and normal hearing in the other frequently complain of difficulty in understanding speech in noisy environments, in church or the theater, as well as in large groups where conversation moves rapidly from one person to another. Since some of these complaints are not universally present in all persons with a severe unilateral hearing loss, it is reasonable to conclude that the small quality differences at one ear and the accessory cues afforded by head movement are sufficient to localize complex auditory stimuli for some people.

The monaural hearing aid usually makes it possible for the bilaterally hearing-impaired person to hear and discriminate the vowel and consonant elements of normal conversational speech. As a consequence, he is able to function more adequately in a hearing world even though he frequently presents those complaints common to the person with a severe unilateral hearing loss in difficult or unusual listening situations. The monaural hearing aid, then, may be considered to be one step in the prosthetic treatment of a hearing loss. Even though the monaural hearing-aid user still may have some problems peculiar to the use of only one ear, he functions more adequately than if he had no amplification whatsoever.

It seems obvious that in order to overcome the complaints of the monaural hearing-aid user a binaural hearing aid should be the answer. With the use of both ears, it frequently has been implied, the bilaterally hearing-impaired need no longer complain of difficulty in understanding speech in noise or following group conversation that moves rapidly from one person to another.[7,16,20] Since he presumably has the effective use of two ears instead of only one, it has been suggested that the intensity, time, and differences in spectral composition at each ear should provide the additional cues necessary for a more reasonable approximation of the normal-hearing person.

This argument for binaural hearing aid use is very compelling, but it ignores the fact that in the normal listener both ears are usually functionally equivalent at both threshold and suprathreshold levels. It would appear that to achieve the advantages of binaural hearing a comparable output from each ear should be present throughout the useful audible range of frequency and intensity. If the output from each ear is different, however, then the utility and presumed advantages of binaural hearing may not be effected.

This concept may be most readily visualized in terms of the phenomenon "binaural fusion," where in-phase sounds of equal intensity appear to be in the center of a listener's head. As the intensity or time relationship between the sounds to each ear is changed, the phantom image of sound appears to move from one side to the other within the listener's head.[31] This fusion phenomenon can be achieved by most persons with almost any kind of auditory stimuli, under headphones as well as in a sound field. Even though some people appear to be unable to experience binaural fusion, it seems reasonable to presume that it has some relation to the effective utilization of both ears, since it represents their coordinated use as well as the inherent capabilities of the person to respond.

If the assumption is valid that binaural fusion is in some way related to effective binaural listening, then the principle should be applied to the hearing impaired when attempting to provide them binaural hearing

with a separate hearing aid for each ear. This means that both threshold and suprathreshold differences between the peripheral hearing mechanisms of the two ears become increasingly important.

IV

Some of the factors which illustrate some of the difficulties involved in applying binaural listening to the hearing-impaired now will be considered. The contribution of each factor to adequate binaural functioning of the normal and the hearing-impaired is not well defined, but the fact that impaired ears frequently are not equivalent in all factors raises issues which do not appear to have been given serious consideration. Some of these factors may not have any significant contribution to effective binaural listening, while others may be the controlling influences. Furthermore, a deficiency in one area may be compensated for by a related function.

Those factors which exemplify at least some of the significant parameters deserving closer inspection may be conveniently divided into three separate areas, as follows: (1) those factors related to threshold levels, (2) those factors related to suprathreshold levels, and (3) those factors related to the ability of the person to utilize vastly dissimilar sound patterns from each ear. Each of these areas will be considered in relation to binaural hearing in hearing-impaired persons.

Threshold Factors

Although threshold listening is not a frequent occurrence in the acoustic environment of most people, there are some interesting factors which may be of some importance. There is always the implication that whatever differences occur at threshold are reflected by similar measures at suprathreshold levels. It is of interest to inspect some of the various monaural threshold factors to see how they may relate to binaural activity.

Pure-Tone Threshold.—Among normal-hearing persons, the hearing level at each frequency for one ear is equivalent to the hearing level at identical frequencies for the other ear. Stated differently, the physical intensity to reach threshold at each frequency in the audible range for normal ears is equivalent. This means that the threshold contour of any one normal ear is relatively equivalent to any other. In other words, the frequency distortion for one ear of a normal-hearing person is reasonably identical to that of the other ear.

In the hearing-impaired, the pure-tone threshold at each frequency on one ear may be vastly different from the threshold at identical frequencies on the other ear. In other words, the threshold contour for one ear will be different from that for the other ear. This may be extremely important when attempting to balance the ears by means of differential amplification. The fact that such amplification would require extremely complex filtering and attenuation networks to "mirror" individual hearing-loss contours makes its clinical application unreasonably complex. With equal amplification on each ear, such as that provided by a hearing aid, it follows that each ear will, in effect, be responsible for different combinations of frequencies and intensities.

The advent of binaural hearing aids, coupled with the fact that the hearing-loss contour on one ear may not be identical to that on the other, raises the issue that possibly an "amplification mirror" of each hearing-loss contour might be more appropriate. This approach may be more profitable for some persons, even though it was found to be generally untrue for monaural amplification.[9]

The frequency distortion inherent in each ear of persons with a bilateral hearing loss brings into focus one of the major differences between monaural and binaural hearing of the normal-hearing population and that of the hearing-impaired population. It is a problem which is ever present and must be considered when the attempt is made to balance the ears to any acoustic stimulus other than a single pure tone. Any

type of complex stimulus near threshold will, of necessity, have a different quality associated with it on each ear of the hearing-impaired person because the response to the frequency-intensity potpouri on each ear will not be the same.

Speech Threshold.—The speech threshold, especially when applied to the hearing-impaired, also reflects the frequency-distortion problem. The relationship between the average of the pure-tone thresholds in the midfrequency region (500-1,000-2,000 cps) yields a fairly consistent estimate of the speech-reception threshold for spondee words among normal-hearing persons or those with a relatively flat hearing loss. On the other hand, those persons with precipitous hearing losses frequently do not yield a consistent estimate of their respective thresholds for speech because the frequency distortion of these impaired ears detrimentally affects the estimate.

When the threshold for speech is measured binaurally, it is difficult to conceive of a binaural effect among all hearing-impaired persons comparable to that observed in normal-hearing persons. On the other hand, some hearing-impaired people should obtain a gain equivalent to the normal listener, while others, under similar circumstances, may experience no benefit. There may be still other hearing-impaired persons in whom the binaural gain is markedly superior to that observed in normal listeners.[7]

Additional Threshold Factors.—Some additional factors which seem to affect threshold determination are the tone-decay phenomenon,[5] the atonal gap,[26] and tinnitus. These factors may contribute to the total management of a case. Tinnitus, for example, is frequently masked by a binaural hearing aid, making it more desirable for those persons where it is a primary complaint. The specific contribution of these threshold factors to binaural and, in some instances monaural, activity awaits further investigation.

Suprathreshold Factors

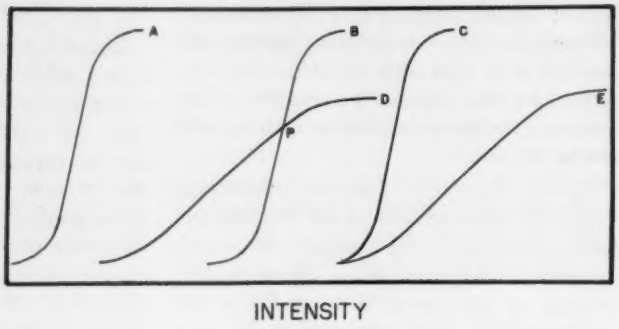
Reasoning from the behavior observed among normal-hearing persons to that observed in the hearing impaired is subject to additional pitfalls when suprathreshold factors are considered. With threshold factors, the primary concern is usually with an operationally defined "point." Suprathreshold measurement brings in the additional concept of *range of function.* It is assumed that the practical clinical concern at suprathreshold levels should be how well a person performs over a wide range of intensity, not just his "maximum" performance at some arbitary level above his threshold.

An additional pitfall in reasoning is related to the assumption that those differences observed between each ear of a bilaterally hearing-impaired person at threshold are necessarily reflected at suprathreshold levels. For example, this most certainly is not the case in those ears exhibiting loudness recruitment, where the responses obtained at high intensities are frequently comparable to those observed in the normal ear.

Along with the concept that a primary concern is range of function, then, is the fact that in some cases differences between the impaired and the normal ear cease to exist at high intensities. This leads to the conclusion that the *rate* of change of function is different for the normal and some impaired ears. Furthermore, there is reasonable doubt that the rate of change of function is identical for two different impaired ears.

This concept of rate of change becomes increasingly important when considering a binaural hearing aid. A basic assumption in effective binaural hearing aid use is to achieve a "balance" between the ears. Notwithstanding whatever nuances are attached to the word "balance," it is apparent that the concept of rate of change has not been given sufficient consideration.

The rate of change of function may be visualized most readily by considering the psychometric function of a particular supra-



Hypothetical normal and abnormal psychometric functions relating the per cent correct to the presentation level of a set of stimuli.

threshold factor. For purposes of illustration, a set of hypothetical psychometric functions relating the per cent-correct response to the presentation level of a set of stimuli is included in the Figure. Curve *A* represents the hypothetical behavior of a normal-hearing person to some test, say speech-sound discrimination as measured by the Harvard phonetically balanced word lists. Curves *B* and *C* represent hypothetical curves for impaired ears whose function is simply displaced to the right. Under ideal amplification conditions, it should be possible to bring both of these functions back to approximate the theoretical normal (Curve *A*), or at least to approximate each other. If Curve *B* represents one ear and Curve *C* the other ear on the same person, it then seems possible to speak of a balance between the ears.

Curves *D* and *E* represent a similar set of conditions. They are different, however, from Curves *B* and *C* in two respects. First, the rate, or slope, of change is different, making the range of intensity greater from where a response is first obtained to the maximum possible score. Second, the maximum percentage of correct responses in Curves *D* and *E* is less than for *B* and *C*, as well as for the theoretical normal. Again, under ideal amplification conditions, it should be possible to bring these two functions together. However, it is not reasonable to conclude that they would ever approximate the theoretical normal.

To recapitulate, there are two pairs of functions represented by Curves *B* and *C* and Curves *D* and *E*. Each member of these pairs, presumably, can be made to approximate each other by suitable differential amplification. If each member of a pair were derived from the impaired ears of separate persons, then, theoretically, the ears would be balanced for the material these functions represent.

Unfortunately, it is difficult to assume that a psychometric function (e. g., for speech-sound discrimination) for one impaired ear on a particular person will be identical to that obtained for the same function on his opposite ear. Such a condition is represented by Curves B and *D* or Curves *C* and *E*. For purposes of illustration, let Curve *C* represent one ear and Curve *E* the other ear of a bilaterally hearing-impaired person who is a prospective candidate for binaural amplification. Under ideal amplification conditions, it should be possible to bring Curve *E* to Curve *D* and, likewise, Curve *C* to Curve *B*, but what about a balance between the ears? It seems reasonable to hypothesize that a balance is achieved only in the regions where these two functions coincide. This is represented by Point *P*. When the presentation level is varied either below or above this *coincidence area*, then the ears would be no longer balanced. It is a matter of conjecture as to whether the shape of the binaural function can be derived from the independent monaural functions and still maintain a balance. There are undoubtedly limits within which the particular function for one ear can vary from that of the other and not

seriously impair any balancing attempted by differential amplification.

Briefly, the question is raised whether it is possible to achieve a balance between ears on all hearing-impaired persons by means of differential amplification. In addition, it seems probable that if a balance is obtained at one intensity level, it may be lost at another, making binaural hearing-aid use extremely impractical and disconcerting for some hearing-impaired persons.

There are many suprathreshold factors which may or may not be uniquely related to binaural listening among the hearing-impaired. Considerable latitude may be permissible for some and not for others. A few of these suprathreshold factors will now be considered which may either assist or act as deterents to adequate binaural listening.

Speech Functions.—The ability of the normal-hearing person to repeat monosyllabic words which sample the vowel and consonant elements of the English language has been used extensively in clinics throughout the country, both for clinical diagnostic purposes and for the evaluation of the efficiency of different hearing aids or hearing-aid systems.[6,30] It is common practice, however, not to construct a complete presentation level versus per cent-correct function for one person but to estimate his probable maximum efficiency (the so-called PB Max). This practice probably has been based on the underlying precepts of a method for estimating the social-adequacy index,[18] which assumed that the slope of the psychometric function for PB words in the person with a discrimination loss was identical to that observed among normal-hearing persons but reached its ceiling earlier.

This presumption has led to the clinical procedure of estimating the maximum-discrimination score at some arbitrary level above the speech-reception threshold for spondee words. Sensation levels of +25 db. to +40 db. frequently have been used for this purpose. Again, these arbitrary levels appear to be based on the observed behavior of normal-hearing persons.

Various approaches by numerous investigators have shown, however, that in those hearing losses characterized by a sensorineural involvement the presentation level versus per cent-correct psychometric function for the PB test material is not comparable to that observed in the normal-hearing or conductively hearing-impaired person.[11,13] In addition to a decrease in the maximum obtainable score, the intensity range necessary to reach that score is greater.

Sensitive to this factor, many clinicians have chosen some fixed high-intensity level as the place to estimate the maximum obtainable PB score. In view of the previous objection to selecting some arbitrary level above the speech-reception threshold and thereby possibly not obtaining a maximum discrimination score, this second approach appears to handle the problem.

However, there are those hearing-impaired persons whose psychometric function for the PB test material first reaches a maximum which is not maintained when the presentation level is increased further. Instead, the function may drop precipitously. This is not infrequently observed in hearing losses characterized by a sensorineural component.[10,18]

It seems that whatever procedure is chosen to estimate a particular hearing-impaired person's discrimination ability should be based on his entire range of function. Within the confines of current clinical methods, however, it is not practical to construct a complete function empirically because the time required is too great and patient fatigue could seriously confound results.

The clinician finds himself on the horns of a dilemma. On the one hand, if he tests "maximum discrimination" at some arbitrary level (e. g., +25 db.) above the speech-reception threshold, he may err by underestimating a particular person's maximum possible score. On the other hand, if he chooses some higher level to use on everyone,

he again may be in error by assuming that once the maximum score has been reached it will remain there.

Common to each of these approaches is the assumption that the criterion measure must be the person's maximum score. This is questioned. It is difficult to justify the clinical utility of a maximum score in a clinical climate where the aim is presumably to estimate the social adequacy of a particular person, both with and without amplification. Since an evaluation of the rate of change by the test material now available is too time consuming, it seems wise not to operate on some a priori assumption, based on normal-hearing persons, to estimate maximum possible performance for a particular hearing-impaired person but to select a point where it may be reasonably assumed that each will operate. Since there is some commitment to a single measure for reasons of efficiency of time, it seems that this need not be a fortuitous maximum discrimination, but the discrimination at a hearing level having some equivalence to both hearing-impaired and normal-hearing persons. It seems that the greatest face validity in predicting a hearing-impaired person's social adequacy, while using a hearing aid relative to that observed among the normal-hearing population, would be some measure of performance at his *comfortable listening level.*

An alternative procedure would be to consider the criterion of effective use of different hearing aids or hearing aid systems on the basis of the slope and 50% point of the psychometric function for speech-sound discrimination rather than a simple "maximum" score and sensitivity measure.

Returning to the binaural hearing aid, it would seem that when each ear is receiving speech at its comfortable listening level, their combined use should be within the coincidence area previously described (Point *P,* Figure). Use of the comfortable listening level, instead of an arbitrary level above the speech-reception threshold, stems from the assumption that the growth of loudness for speech in each ear may not be equal.[14] A "built in" standard, which appears to have a greater face validity than the PB Max, is the comfortable listening level. Furthermore, it has been found that when each ear is independently set to its comfortable listening level with a binaural hearing aid, sounds in the median place cease to be located at one ear or the other. This result suggests that the hypothesis of a coincidence area is not unreasonable. Under these conditions, it seems possible to say that the ears are balanced, realizing that a speech input above or below the limits of the coincidence area may throw the ears out of balance.

Although this discussion has centered about the psychometric function for the PB test material, the same remarks could apply just as well to the function for spondee material.[17] Furthermore, it has been assumed that once a comfortable listening level for speech has been obtained continued presentation of speech will remain comfortable. In other words, it has been assumed that adaptation for speech does not occur. If it does, then it seems as if binaural-hearing-aid use becomes even more complex than the previous discussion would indicate.

Pure-Tone Functions.—Although the clinical application of amplification is primarily concerned with the more pragmatic measures of speech reception and discrimination, it is of interest to consider some pure-tone factors which may contribute to these speech functions. More specifically, since a speech function may be considered to be derived from various pure-tone factors, they also deserve some inspection.

A suprathreshold measure utilizing pure tones is the loudness function. In one normal ear, the growth of loudness at each frequency is equivalent to that observed for all other normal ears. This is not the case in those impaired ears characterized by loudness recruitment, where the loudness attributed to a given increase in sound intensity is greater than that observed in the normal ear. Moreover, the abnormally rapid increase in loudness at one frequency will not be identical to that observed at other frequencies. If both ears of a bilaterally hearing-impaired person exhibit loudness

recruitment, it is extremely unlikely that they are identical, even at one frequency. Since hearing aids are frequently used by persons exhibiting loudness recruitment, the utility of a binaural hearing aid needs to be viewed from this aspect of probable difference between the ears prior to any type of attempt to balance the ears.

A similar argument can be made for differences between impaired ears in their differential intensity sensitivity, difference limen for frequency, adaptation, and harmonic as well as possible intermodulation distortion. Actually, it appears increasingly remarkable that some people receive any benefit whatsoever from a binaural hearing aid. On the other hand, the fact that one ear may differ markedly from the other leads to the conclusion that some of these factors do not contribute significantly to adequate use of a binaural hearing aid.

Nonperipheral Factors

Aside from the peripheral factors at threshold and suprathreshold levels, there are additional considerations which need attention. Recent investigations dealing with retrocochlear involvements of the auditory system reveal that severe auditory disturbances can occur which are similar in many ways to cochlear involvements but differ in site of the pathological condition.[3,25,29,34] Throughout the previous discussion, it has been assumed that the retrocochlear biological requisites for binaural hearing were intact. When both ears are used, retrocochlear factors become increasingly important. For example, there may be a "squelch" effect, where sudden loud sounds appear to be less disturbing to the listener when both ears, rather than only one ear, are used.[21] Similar effects, such as "summation" of discrimination,[2] greater ability to hear speech in noise,[15] threshold summation,[4,19] and many other factors seem not to be related to the peripheral hearing mechanism alone. Whatever these factors might be, or are related to, it is apparent that when both ears are used slight deviations from normality could seriously impair the effective use of both ears.

V

In conclusion, it appears that the concept and application of binaural hearing aids for the hearing-impaired has been somewhat oversimplified. The purpose of this present discussion is to focus attention on what are believed to be many of the previously neglected aspects of binaural hearing aid use. Although the argument for binaural hearing aids is very compelling, it neglects the fact that the ears of most hearing-impaired persons frequently are not reasonably equivalent. It seems that the relationship between the impaired ears on one person at both threshold and suprathreshold levels as well as possible nonperipheral factors requisite for effective binaural listening need further consideration before it is possible to generate a consistent clinical framework for the application and evaluation of binaural hearing aids.

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Case Reports

Pyocele of the Sphenoid Sinus

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Mucocele is defined as an accumulation and retention of mucus within a sinus as a result of an obstructed outlet. When the contents of the cavity are purulent in character, the condition is referred to as a "suppurating mucocele" or "pyocele."

Skillern [1] set forth several criteria for differentiating mucoceles from pyoceles, which include the rate of growth, inflammatory symptoms, tenderness and consistency on palpation, severity of orbital symptoms, characteristics of the contents of the cyst, and the finding of pathogenic organisms on culture of this material. These criteria are useful in the typical case, but there are numerous borderline instances in which the foregoing criteria are of little aid in differential diagnosis.

When a low-grade pathogenic organism is involved in the development of a pyocele, the rate of cystic growth will not necessarily be appreciably more rapid than that of a mucocele; the patient may not exhibit any systemic manifestations of inflammation, and it will be difficult to ascertain whether or not the cultured organism is a contaminant. Palpation also is likely to be an unreliable criterion, particularly so in the case of the sphenoid sinus, because of its inaccessibility. The literature contains many examples of severe ocular symptoms and even blindness as complications of mucoceles; consequently, the severity of orbital symptoms can be misleading. Although the contents of the cyst are helpful in the majority of instances, when we find thick yellow or green cheesy material the question arises as to whether or not this represents inspissated mucus or is the result of a long-standing, smoldering inflammatory process. In cases in which there has been previous antibiotic therapy, the picture is further distorted. In these borderline cases which we have been discussing it seems important to elicit, when present, a history of nasal discharge. A helpful aid in the differential diagnosis is the establishment, through the history or by direct inspection, of the fact that there is or has been nasal discharge of clear mucoid, mucopurulent, or frankly purulent character. When all factors are considered, however, in certain cases it still will be difficult (or impossible) to say with assurance that the particular cyst is a mucocele or pyocele.

Turner,[2] in 1907, could not find any cases of mucoceles of the sphenoid sinus reported in the literature, and Linthicum and associates,[3] in 1946, estimated that not more than 25 cases had been published in the medical literature. We found, on review of the English literature, that it was 1932 before O'Shea [4] reported the first case, and since then only eight additional cases [3,5-10] of sphenoid sinus mucoceles have been reported.

Experts on the subject of mucoceles agree that a defective outlet is the etiologic factor, but the mechanism behind the obstruction is controversial. The majority of authors favor extension of local inflammation from a neighboring part which causes partial, intermittent, or complete ostial ob-

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struction. However, as pointed out by Turner,[2] and later on Dabney,[11] in a significant number of cases the ostium is patent and seems adequate for normal drainage. It is these latter examples which seem to be logically explained by the mechanism proposed by Tamari and O'Neil[12]; they stated that there is a sinus submucosal injury due to trauma, hemorrhage, or infection with an accumulation of exudate beneath it. As the exudate increases, the elevated mucosa herniates toward the sinus cavity.

Trauma, as the exciting cause of many mucoceles, was championed by Howarth,[13] and in several instances the evidence appeared to be overwhelmingly in support of such an etiologic agent. Dabney[11] drew attention to a mechanism favored by many authors and explained as cystic dilatation of one or more mucous glands, cystic degeneration of the mucosa, or polyps in the sinus.

These theories of etiology all have their conception through study of mucoceles in accessory sinuses other than the sphenoid. Though the hypotheses may hold true for mucoceles of the frontal and ethmoid sinuses, the anatomic, histologic, and physiologic peculiarities of the sphenoid sinus cited by Proetz[14] do not necessarily make them applicable to the sphenoid mucocele. At this time, therefore, we are forced to admit that the etiology of mucoceles in general, and that of the sphenoid sinus in particular, is poorly understood.

When a mucocele of the sphenoid sinus has been allowed to progress to the extent that it has eroded through one or more of its walls, it may give rise to complications elicited through pressure on one of the 13 major structures[14] situated in the immediate neighborhood. The symptoms and signs of a mucocele beyond the confines of the sphenoid sinus for the most part, however, arise from pressure on the structures transmitted to or from the orbital cavity in the superior orbital fissure and optic foramen at the orbital apex. These structures include the oculomotor nerves, trochlear nerves, ophthalmic divisions of the trigeminal nerves, abducens nerves, some filaments from the cavernous plexus of the sympathetic nerves, the orbital branch of the middle meningeal arteries, a recurrent branch from the lacrimal arteries to the dura mater, the ophthalmic veins, the optic nerves, and the ophthalmic arteries. Smith[15] introduced the phrase "orbital apex syndrome" for the clinical manifestations of a lesion which affects the nerves and vessels passing through the superior orbital fissure and optic foramen and recognized the importance of sphenoid sinus disease in manifesting the characteristics of the syndrome.

It is of interest to note the usually long period of symptomatology prior to the final diagnosis in the nine reported cases; the average duration was approximately 7 years, while in individual cases the period ranged from 3 months to 21 years. The long-standing complaints are frequently unilateral headache, anterior and posterior nasal discharge, anosmia, nasal obstruction, nasal polyps, and a history of sinusitis. It is the late complications, when the mucocele erodes the sinus wall and compresses the neighboring structures, however, that usually lead to the correct diagnosis.

The difficulty of diagnosis is attested to by Linthicum and associates,[3] who stated that the diagnosis of sphenoid sinus mucocele has seldom been made preoperatively, and by Neffson,[9] who stated that there have been no roentgenographic reports where the diagnosis was correctly made. The truth of these statements is borne out by the fact that in seven of the nine reported cases the initial diagnosis was neoplasm and in only one case was the neoplasm thought to arise from the sphenoid sinus. In one other case, the diagnosis was aneurysm of the internal carotid artery, and in the ninth case, it was mucocele of the sphenoid sinus.

The commonest late manifestations of sphenoid mucocele are diminished vision to total blindness, diplopia, proptosis, exophthalmos, ptosis, and pain in the eye on the

side of the lesion. One interesting observation is that in six of the reported cases the ocular signs, once established, tended to fluctuate, undergoing remissions and exacerbations. This characteristic, which occurs not infrequently, should be a useful aid in the differential diagnosis of the orbital apex syndrome.

Report of Case

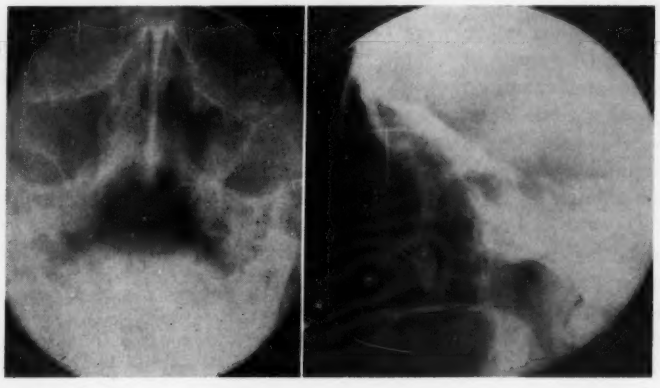
A 58-year-old white woman was first seen at the Mayo Clinic on July 1, 1935, with the complaint of repeated attacks of biliary colic. She also complained of severe headaches, which she described as a dull heavy feeling, in the left supraorbital region; occasionally they were associated with nausea and vomiting. At this time her blood pressure was normal, and the headaches were thought to have been of a migrainous type. A nonfunctioning gallbladder was demonstrated on x-ray examination, and cholecystectomy was performed subsequently.

The patient was not seen again at the clinic until Oct. 30, 1958. In 1948 she had been told that she had hypertension. She now requested a general examination, with particular reference to high blood pressure and frequent headaches. Her blood-pressure determinations averaged between 210 and 240 mm. of mercury systolic and 120 and 135 mm. diastolic, and a diagnosis was made of severe hypertension as manifested by severe hypertensive sclerosis and narrowing of the retinal arterioles with moderately severe focal constrictions. All further studies in regard to her hypertension, including electrocardiogram, roentgenogram of the thorax; determinations of urinary catecholamines, serum potassium, blood sugar and blood urea, and urinalysis, which showed minimal albuminuria, were within normal limits and did not reveal any complications. The patient was then sent to the Section on Otolaryngology and Rhinology for consultation in regard to the headaches.

The patient had been troubled with two varieties of headache for years, the first type being a dull heavy feeling above and behind the left eye, which radiated down the left side of her neck and which had been mentioned during her previous visit to the clinic in 1935. This headache frequently awakened her in the early morning, was associated occasionally with nausea and vomiting, and was always worse during the morning. She thought that the headache first began after an episode of "dust pneumonia," and for the past five or six years, it had become severer and more frequent. The second type of headache had been of long duration as well and consisted of a dull ache in the occipital region, occurring two or three times a week. Both her mother and her sister had been treated elsewhere for migraine headache.

In 1948, she became aware of a bad odor, which she compared to the "smell of a dead cat," in the left side of her nose. This smell fluctuated in intensity and frequently disappeared for long periods. On examination of the nose, purulent material was seen between the nasal septum and the left middle turbinate, and roentgenograms of the sinus showed an opacity in the left sphenoid sinus which the radiologist interpreted as a pyocele (Figure). The patient was afebrile; the leukocytes numbered 6,100 per cubic millimeter of blood, and the sedimentation rate was 14 mm. in an hour (Westergren method). After the introduction of cocaine and epinephrine nasal packs, it was possible to obtain, by suction, some cheesy purulent material from the region of the ostium of the left sphenoid sinus.

The patient was hospitalized, and on Nov. 5, 1958, under local anesthesia, the left sphenoid sinus was explored through a transnasal approach. A small polyp was removed from the anterior surface



Two views showing marked density of bone surrounding the left sphenoid sinus.

of the sphenoid, and the natural ostium, which was patent, was exposed. Through the use of a suction tip, foul-smelling, granulated, purulent material was obtained from the sinus. The entire anterior wall of the sinus was removed, revealing a cavity filled with thick yellow cheesy material and lined by a moderately thickened membrane which shelled out like a sack. The walls of the sinus appeared slightly thickened rather than thinned, which was unusual in view of the long history of symptoms but presumably was explainable on the basis of bone reaction to inflammation. A culture of the material from the sinus grew Hemophilus influenzae and Staphylococcus of undetermined species, and microscopic examination of the material showed acute inflammatory and necrotic tissue.

The patient's postoperative course was uneventful, and the use of chlorothiazide (Diuril), hydralazine (Apresoline), alseroxylon (Rauwiloid), and pentolinium (Ansolysen) was started for her hypertension. After eight days of therapy, her blood pressure fell to levels averaging 160 mm. of mercury systolic and 90 mm. diastolic in the sitting position and 140 mm. systolic and 85 mm. diastolic in the standing position. The patient was dismissed at this time with almost complete freedom from headaches.

Comment

The possibility of mucocele or pyocele of the sphenoid sinus should be stressed in the differential diagnosis when there is a history of unilateral headache of long standing associated with nasal complaints; when headache prompts the acquisition of roentgenograms of the skull which show erosion of the sphenoid bone; when the orbital apex syndrome is evident, and particularly when the signs are subject to fluctuation in severity. If the possibility of a mucocele of the sphenoid sinus is entertained in the differential diagnosis, roentgenograms of the sinus should be made and examined carefully, and in doubtful cases a transnasal investigation of the sphenoid sinus should be undertaken; these procedures aid in making a diagnosis. The importance of diagnosis is obvious from the standpoint of prolonged discomfort to the patient, avoidance of the complicating orbital apex syndrome with its ultimate serious sequelae, and avoidance of unnecessary surgical measures and x-ray therapy.

Summary

Although there have been nine cases of mucocele of the sphenoid sinus reported in the English literature, we could not find a previously reported case of pyocele of the sphenoid sinus.

The etiology of mucoceles and pyoceles of the accessory sinuses is poorly understood, and although the theories of etiology may hold true for mucoceles of the frontal and ethmoid sinuses, they are not necessarily applicable to the sphenoid mucocele.

Mucoceles of the sphenoid sinus are usually not diagnosed until complications have arisen through pressure on one of the major structures situated in adjacent regions. A frequent late complication that is useful in differential diagnosis of mucoceles is the orbital apex syndrome, which is commonly subject to fluctuation in severity.

In certain borderline cases it is extremely difficult to differentiate between mucoceles and pyoceles, and when a nasal discharge is present or has been present, it is important to note its character by direct inspection or through the history.

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Rhabdomyosarcoma of the Naso-Orbital Angle

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Rhabdomyosarcoma is an uncommon tumor. Until recently many cases were not diagnosed as such, histopathologically, because no cross striations in the cells were evident. Stout,[1] in 1946, described the various appearances of tumors arising from skeletal muscles and made it possible to classify these tumors, despite the lack of demonstrable cross striations.

Rhabdomyosarcoma, in contrast to other sarcomas, tends to disseminate via the lymphatics. In the later stages, however, spread occurs through the blood stream.

Histologically, the appearance often suggests that of an anaplastic carcinoma or that of a fibrosarcoma; the cells may be of many types—long, short, spindle cells, or ovoid cells with one or two nuclei. There may also be some giant cells (Nanavati[2]).

As far as treatment is concerned, surgery or surgery combined with radiotherapy has been used. However, the results are not encouraging. Delcourt and Dustin[3] have mentioned the use of nitrogen mustards.

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From the E. N. T. Department, Dar el Chifa Hospital.

Fig. 1.—Photograph of patient, showing tumor.

Report of Case

A 6-year old child was brought to the outpatient department in June, 1958, with a mass on the left side of the face. The mass had been there for six months; there was no history of trauma, and it had been progressively increasing in size—more rapidly during the past three months. There were no other complaints. Past history included admission to a charity hospital, prior to the appearance of the mass, for removal of what was described as a nasal polyp.

Examination revealed a young child in no acute distress with a mass in the left naso-orbital angle. The mass was round and firm in consistency, with a diameter of 1¼ in. The skin over the mass was red, stretched thin, and attached to it; the tumor was fixed to the underlying structures and was pushing the inner canthus upward and the root of the nose toward the other side (Fig. 1). Both sides of the nose were free. The ear canals and tympanic membranes were normal. The tonsils were present and not enlarged. Several glands

Fig. 2.—Occipitomental view, showing soft tissue mass.

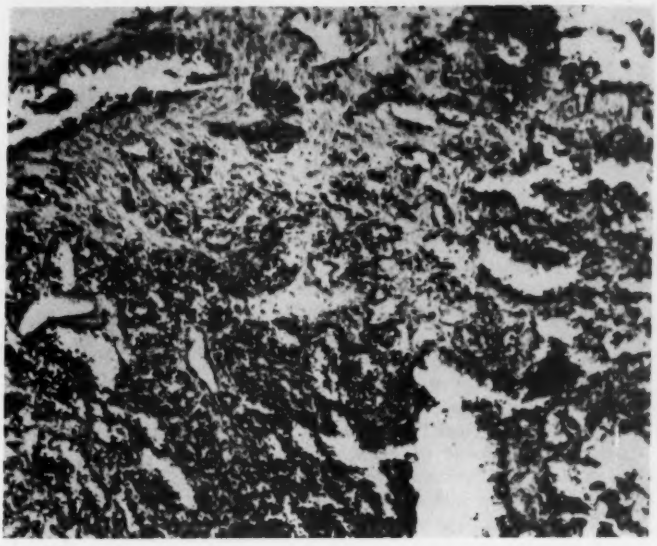

Fig. 3. — High-power photomicrograph of tumor.

were palpable in the left submaxillary and jugulodigastric areas; these glands were firm and movable.

X-rays in the Waters' and anteroposterior positions revealed the maxilla and orbital margins to be intact. The sinuses were free. It was felt that the tumor was of soft tissue origin (Fig. 2). A chest x-ray was negative.

Routine laboratory tests were not remarkable.

Under general endotracheal anesthesia, an incision 2 in. long, parallel to and below the left mandibular margin, was done; an enlarged node attached to the underlying submaxillary gland was dissected out with the salivary gland, and the wound was closed with drainage.

The pathological report was that the tumor was definitely malignant and that its picture was very strongly suggestive of an embryonal reticulated type of rhabdomyosarcoma (Fig. 3). The parents were advised to have the child submitted to deep x-ray therapy, but they refused any treatment.

Summary

A case of rhabdomyosarcoma of the naso-orbital angle is presented. Such tumors are quite rare and represent a challenge from the points of view of both diagnosis and treatment.

4, Baehler St.

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Pyogenic Granulomas

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Two patients recently brought the entity of pyogenic granuloma to my attention.

A young man, 24 years of age, stated that he had had bleeding from his tongue for the past week. He had not noticed anything on his tongue until two weeks previously when he saw a small white nodule there. On examination, he showed a brownish-red mass about 8 mm. in diameter, attached to the midline of the tongue about one-third of the way back from the tip. When a snare was put around the mass, it appeared to have a thin stalk and was removed easily. The base was cauterized with a silver nitrate applicator.

The report from the laboratory stated that it was a pedunculated lesion covered by stratified squamous epithelium, with masses of endothelial cells containing many capillary spaces beneath the epithelium. Between the cellular masses were acute inflammatory cells with areas of loose basophilic connective tissue. The diagnosis was "pyogenic granuloma."

A young man of 23 stated that for the past two weeks he had had nosebleeds off and on. A tumor, reddish in color, was removed with a snare from the floor of the nose on the right side and the base cauterized with a silver nitrate applicator. On removal, there appeared hardly any indication of where the tumor had been attached by a thin stalk. The report by Dr. C. B. Preacher was similar to his report on the previous case.

Two months later the patient again had a mass of the same size as originally seen in the same area and reported that he had had more nasal bleeding. The mass was removed, and this time the base was cauterized with electrocautery. The report again showed pyogenic granuloma, and the patient had no further recurrences.

Comment

In a review of the literature on pyogenic granulomas, no articles were found in ear, nose, and throat publications and it was felt that perhaps this entity is not as well known by E. N. T. men as it should be. A large part of the reports seen were either in dermatological or dental journals.

Submitted for publication Dec. 15, 1958.

A good idea of the frequency of this lesion was given in a paper by Kerr,[1] in 1941. He collected 289 cases from the surgical material submitted to the department of pathology at the University of Michigan in 25 years. Of these 289 there were 143 in the oral cavity (20 on the tongue) and 21 in the nares. Thus it is something that might be seen by an otolaryngologist.

Kerr stated that, in 1870, Bollinger and, in 1884, Rivolte described botryomycosis in horses after castration. In 1887, Poncet and Dor described a lesion in man comparable to that described by Rivolte and designated it as "botryomycosis hominis." Reports in German literature questioned the etiology and terminology. Hartzell, in 1904, and Wile, in 1910, first reported it in American literature.

Riley[2] states that it is seen commonly in dermatological practice. It is a rapidly growing granulomatous lesion that usually occurs at the site of an injury. It is usually less than 1 cm. in diameter and usually moist, fleshy, mushroom-like, and bright red, bluish-red, or brownish-red. It occurs frequently on the hands, arms, and face but may occasionally occur on mucous membranes. The typical histologic picture is that of an infected angioma. It is typically pedunculated, showing numerous capillaries and newly formed vessels, densely infiltrated with polymorphonuclear leukocytes.

According to Rupe and Lockward,[3] the lesions bleed on slight provocation and are most likely to develop in locations exposed to trauma. The tumor develops rapidly and persists for months unchanged. If incompletely excised it quickly recurrs. Pure cultures of Staphylococcus pyogenes have been repeatedly cultured from these lesions.

Satisfactory treatment in the past has been excision and cautery.

The most interesting aspect of the cases I have seen is how typical they are of pyogenic granuloma and how little known such a frequent condition can be. Probably it would appear to be more frequent if all "polyps" were studied histologically.

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Papillomas of the Nose

ROBERT B. TRUCKEY, M.D., Seattle

Historically, the term papilloma was introduced into the literature in the mid-19th century by Kramer.[1] Hopmann,[2] in 1883, specifically called attention to the occurrence of hard and soft types of papillomas, and at that time, the soft types were associated with inflammation and the hard types were regarded as true tumors.

It was also noted by other observers that the epithelium preponderated in the hard variety and the stromal elements in the soft, or inflammatory, type. Kiesselbach,[3] in 1893, described a case of papilloma of the nose that eventually turned cancerous, and since that time other cases have been reported in the literature of the so-called hard type papillomas that have degenerated into cancer. Kramer and Som,[4] in a review of the literature in 1935, could find only 86 cases since 1856 of this rare type of tumor. Ringertz,[5] in 1938, described nine cases of true papillomas and gave a complete description of the microscopic anatomy. He emphasized the inverted character of nasal papillomas, i. e., how the epithelium grows downward into the stroma and in this way breaks up the stroma. This is in contrast to the behavior of other papillomatous formations, such as cutaneous warts, in which the epithelium in proliferating draws the stroma into lobes. This growth pattern of inverting character is important because it can cause pressure necrosis of tissue and bone if of sufficient size. Certainly, this type of tumor tends to be locally invasive of contiguous structures and may involve an entire unilateral sinus system.

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Read before the Section on Laryngology, Otology, and Rhinology at the 107th Annual Meeting of the American Medical Association, San Francisco, June 27, 1958.

The *incidence* of this type of papilloma is rare. However, it is felt that the lesion has not found its way into the literature more often because of its resemblance to common polyps and, therefore, is simply not recognized. In the same aspect, many authors feel that polyps should be sectioned, especially if they have recurred frequently in the past.

Men are more frequently affected than women with this lesion. The usual age of onset is between 40 and 50 years. The commonest site is the ethmoid area and secondarily the antrum.

Etiology of this tumor is unknown at present. However, Jarvi,[6] in 1944, found inclusion bodies in nasal papillomas and suggested that viruses might be a possible factor. A similar hypothesis has been advanced by Eggston and Wolff [7] and other authorities.

Grossly, papillomas vary in appearance. They may be red or gray in color, lobate or sessile in character. The consistency may vary from soft, limp, and friable to considerable degrees of firmness, approximating that of a hard-boiled egg.

Microscopically (Figs. 1-3), there is a metaplasia of epithelium and an inward growth of the epithelium forcing the stroma apart. This results in the formation of crypts. There is always a definite basement membrane, and the stroma is rich in blood vessels. Inflammatory cells are scattered throughout the stroma. In addition to metaplasia, the epithelium also undergoes papillary hyperplasia. Surface epithelium can be squamous, columnar ciliated, or transitional; cellular activity and mitotic figures are variable.

The *symptoms* are those depending on the size and location of the growths. Gen-

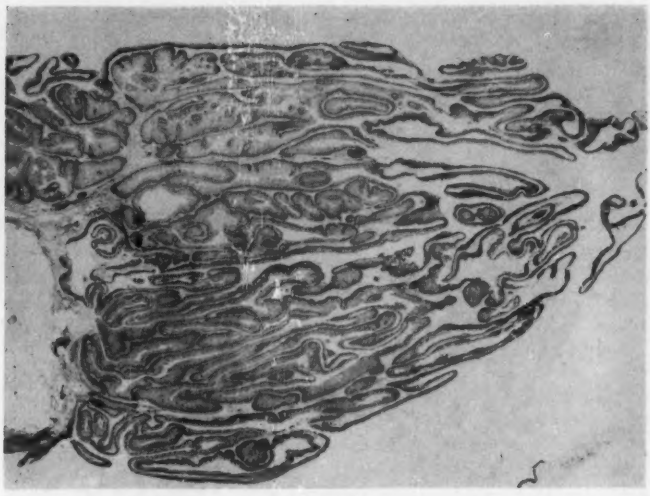

Figure 1

erally, the lesions are very extensive by the time they cause symptoms. They may cause nasal obstruction, loss of smell, and nasal discharge. Epistaxis is fairly common and may be present early in the onset of the tumor growth. If there is pressure necrosis of bone, the disease may cause orbital symptoms, headache, or pain in the teeth. Inflammation in the sinuses caused by blocking and stasis is generally present and accentuates the symptoms. Occasionally, the nasolacrimal duct will be occluded. Over the years, in the reported cases, the incidence of recurrence is very high. Also, the tendency to malignancy is an accorded fact and should be borne in mind when the initial surgical step is anticipated.

The *treatment* has gradually evolved from repeated local excision to more radical steps, at present. A considerable number of these tumors were treated in the past with radiation therapy, either radium application or

Figure 2

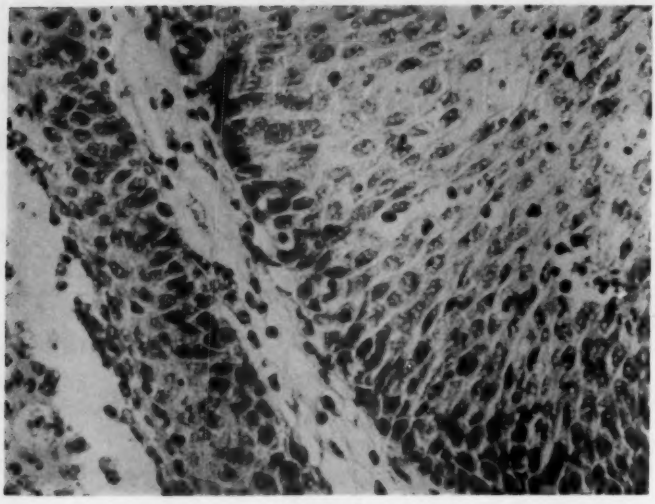

Figure 3

x-ray. This form of treatment has proven of little value. Also, most authors have felt that surgery has not been radical enough in previous years. Certainly, most of the cases are far advanced, and if they show signs of inverting quality of the papilloma and pressure necrosis of bone, nothing short of rhinotomy and radical sinus exenteration will suffice.

Report of Cases

Case 1.—This 60-year-old white man entered the hospital with a history of nasal obstruction and polyps for 10 years. In the 10-year period, the patient had four polypectomies and other treatment, mainly for chronic nasal discharge. Past history was otherwise not remarkable. His physical examination was essentially normal, except that his nose showed a marked humping of the bony pyramid on the left side, which was crepitant by palpation. There also was a small cystic fluctuant mass near the medial canthus of the eye. In the left nasal cavity, there was a large, meaty, reddish type tumor and several smaller polypoid type masses with excessive mucopurulent discharge. The tumor mass extended posteriorly into the choana. X-ray findings are given in Figure 4. There was marked deformity of the left nasal bones which seemed to be associated with soft tissue swelling of the left nares area, displacing the septum to the right side. There was a bilateral antral clouding with accompanying density in the ethmoidal cells. The frontals appeared clear. The patient was taken to surgery, and a polypectomy revealed the whole ethmoid area on the left to be involved. The histological picture revealed epithelial papilloma of the inverting type, and after this examination the patient sustained a radical frontoethmoidectomy through a Killian Lynch incision. Within two months, however, the patient returned because of a swollen left eye and with signs of cellulitis. This cleared with treatment, and frontonasal passage was secured with a large catheter. The patient was followed for another five months, and rebiopsy at that time showed recurrent papillomata. The patient then had a left lateral rhinotomy; removal of the left nasal wall, ethmoids, and sphenoid, and left frontal sinus ablation. The patient has been followed for some four years with no sign of recurrence to date.

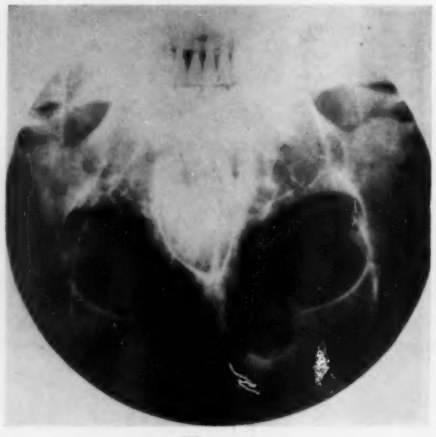

Figure 4

Figure 5

CASE 2.—This 50-year-old white man had a history of right-sided nasal obstruction for 30 years. The patient had had a submucous resection and polypectomy 30 years previously as well as repeated polypectomies in 1930 and 1950. In the past six months before his hospitalization, he was completely occluded on the right side and partially on the left. He had considerable constant mucopurulent discharge. The right nasolacrimal duct was also occluded. His past history was not remarkable. Physical examination was essentially negative, except for the nasal findings. The left nasal cavity was partially obstructed because of a septal deflection, and the right nasal cavity was occluded with a large reddish tumor mass. Biopsy revealed epithelial papilloma. X-rays (Figs. 5 and 6) of the sinuses showed homogenous opacification of the right antrum, and continuous to this sinus, there appeared a density on the lateral side of the nasal cavity. There also was some clouding of the right ethmoid sinuses and the right frontal sinus. Detail was not sufficient to determine if there was any bone destruction affecting the medial sinus wall or elsewhere. The patient was taken to surgery, and a right-sided rhinoplasty approach was used. The incision was made above the eye along the lateral nasal wall down to the columella, and an internal septal incision was made. The upper lateral cartilages were freed from the septum. The lower lateral cartilages were separated at the dome. The entire right side of the nose was elevated after an external osteotomy was performed. This afforded good visualization of the nasal cavity. It was noted that the lateral bony wall was missing owing to bony erosion from the antrum, and the entire area was cleaned of a large block of tissue. The eye was then retracted laterally; the anterior ethmoidal artery was ligated, and the ethmoids were exenterated; the frontal on the right side was clean—however, the mucosa was removed. The sphenoid on the right side was opened, and tumor tissue was present. This was removed entirely. The patient has had a normal postoperative course and has had, at present, two years with no sign of recurrence.

CASE 3.—This 42-year-old Filipino man, a laborer, was admitted to the hospital because of a tumor in his right nasal cavity, swelling of his right lower lid, and malar prominence. His his-

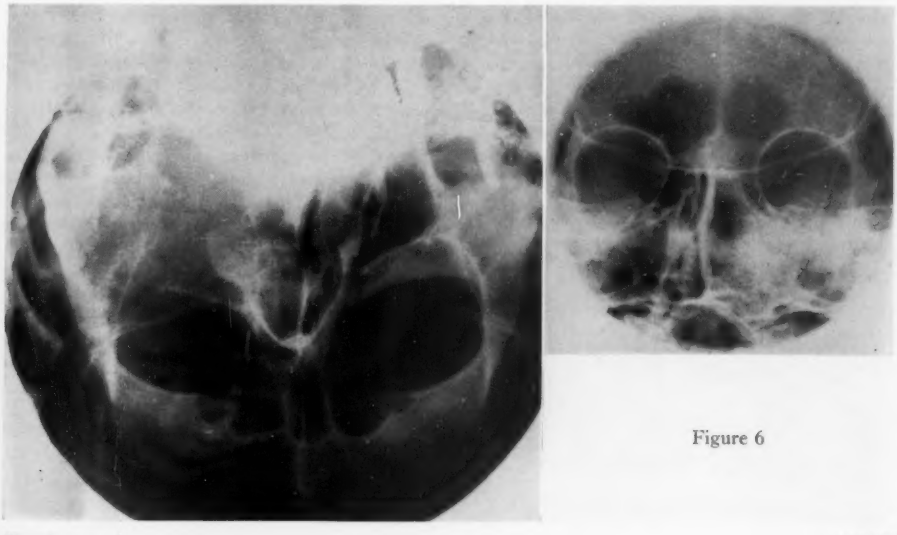

Figure 6

tory was of approximately three months' duration. The swelling was painless, and his right nasal cavity was obstructed. He occasionally had blood-tinged nasal discharge. Past history was not remarkable, nor was his physical examination. There was a fullness of the right malar prominence and a small draining fistula in the infraorbital ridge laterally beneath the external canthus. His left nasal cavity was clear, but the right nasal cavity contained a papillary tumor, soft and reddish, occluding the entire side and extending into the nasopharynx. Biopsy revealed epithelial papilloma, inverting type. X-rays (Fig. 6) showed a soft tissue mass in the right antrum with thinning of the bone superiorly and medially. The tumor board recommended wide surgical excision and probable exenteration of the orbit because of the tendency to recur and the possibility of malignancy. The patient was taken to surgery. A tracheotomy, bilateral ligation of the external carotid arteries, radical antrotomy, and ethmoidectomy were performed. The approach was made by splitting the lip laterally along the alar nasal fold at the side of the nose to the inner canthus and then laterally to the outer canthus infraorbitally. The antrum was entered and found to have a fungating mass filling the entire antral area. The lateral wall of the sinus was removed and it was found necessary to take the palate on that side (because of involvement) as well as the orbit and its contents. The ethmoid cells were completely exenterated, also because of involvement. The frontal sinus was free of disease. Split-thickness skin grafts were removed from the abdomen and applied to the orbital area. During the postoperative course the patient had a dental prosthesis made, and repeated biopsy specimens from suspicious areas have revealed no recurrence. At present, this case has been followed for six years.

Conclusion

Three cases of nasal papilloma are presented. The characteristic microscopic appearance and clinical aspects of these rare tumors are given. The size, tendency toward recurrence, and possibility of malignant change are such that radical surgery was required initially.

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Clinical Notes, New Instruments and Techniques

Dry Treatment of Infected Mastoid Cavities

JAMES L. SHEEHY, M.D., and HOWARD P. HOUSE, M.D., Los Angeles

The chronically or intermittently discharging mastoid cavity seems to be a problem without solution at times and has discouraged some from advising or undertaking mastoid surgery. It is a rare otologist who does not have at least a few problem cases of this type that have resisted all forms of therapy, including curettage and skin grafting. It is the purpose of this paper to point out the underlying factors which tend to prolong the infection and to outline the dry (powder) treatment of infected mastoid cavities.

Although many factors enter into any infection, there are three main factors underlying all mastoid cavity infections:

(1) Moisture
(2) Presence of opportunists (bacteria and fungi)
(3) Debris and diseased tissue

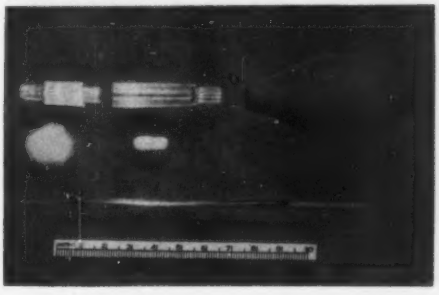
Many bacteriocidal and fungicidal ear drops have been recommended for the treatment of cavity infections. Usually these medications are very effective in reducing the amount and in controlling the odor of the discharge. Frequently, however, the cavity has continued to drain or has remained moist, and malodorous discharge recurrs after the drops are discontinued because moisture has *not* been eliminated. As a direct result of this moisture and its effect on the underlying epithelium, the cavity frequently fills with debris.

The principle of treatment of an infected mastoid cavity is to eliminate the three underlying factors. 1. The cavity must be cleaned of all debris. This can best be accomplished by gentle spot suction and the use of small cotton applicators. It is important that the otologist takes the time to inspect and clean all areas, paying particular attention to the tip region and the area immediately above the meatus. These areas are often difficult to see and, as a result, are frequently neglected. All *infected* granulation tissue should be removed with a forceps or lightly cauterized with a caustic such as 10% silver nitrate. 2. After all debris has been removed, the entire cavity is dried with a cotton applicator, with the use of a blotting rather than a wiping action. All sheets of fungus and accumulated pus are removed in the process of cleaning and drying. 3. After the ear cavity has been thus prepared, an antibiotic powder, consisting of equal parts of chloramphenicol and sulfanilamide, is gently insufflated. This combination has been used because of its broad antibacterial spectrum and because it does not "cake" in the cavity or in the insufflator, as do tetracycline derivatives. Chloramphenicol alone is used if the patient is sensitive to sulfonamide drugs.

Daily treatment as outlined above is indicated. In certain cases, particularly in children and uncooperative adults, this should be done in the office, but usually the patient will be able to carry on treatment at home.

Complete indoctrination of the patient in the principle of treatment and the technique of cleaning and powdering his cavity is of utmost importance and requires time, on the part of either the otologist or his nurse. Aural hygiene is discussed in detail. (The patient is advised to place a large piece of cotton in his meatus and cover it with petrolatum before showering or washing his hair.) A fine wire applicator is dispensed, and instructions are given in the technique of wrapping the cotton on it. A bend is made in the end of the wrapped applicator to facilitate its use and instructions are given in the technique of drying the cavity. A blotting rather than a wiping motion

Fig. 1.—Armour Inhalator and wire applicator, disassembled.



Submitted for publication Feb. 23, 1959.

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From the Otologic Medical Group and the Department of Otolaryngology, University of Southern California School of Medicine.

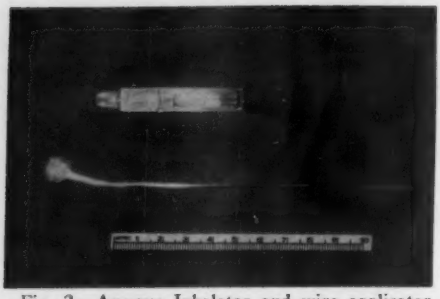

Fig. 2.—Armour Inhalator and wire applicator, assembled and ready for use.

is used to prevent the patient from traumatizing the tissues. The cavity should be dried two or three times a day, depending upon the amount of discharge. After each cleaning, one or two puffs of chloramphenicol-sulfanilamide powder is insufflated with the use of an Armour Inhalator. A No. 4 capsule, containing 50 mg. of each antibiotic, is used in the inhalator and is placed with the open end, toward the bulb. (The instructions with the inhalator direct just the opposite.) One capsule should be sufficient for 10 to 20 treatments. The patient is seen in the office in four to seven days. Frequently there will be no evidence of moisture, even after this short treatment period. The patient is seen again in about three weeks, at which time any crust can be removed. The importance of aural hygiene is again reveiwed at this time, and the patient is given a three-month appointment for routine cavity hygiene. If any moisture should develop in the meantime the patient should reinstitute treatment and come into the office if discharge persists more than three days.

This dry (powder) treatment has been used by one of us (J. L. S.) for three years with great success. It has been adopted by our entire otologic group, and the results have at times been dramatic, particularly in long-standing problem cases. Its use is equally effective in certain cases of subacute and chronic otitis externa and otitis media, where the same underlying factors are present. This is especially true in the acute flare-up of a chronic otitis media with central perforation, where the disease is limited to the middle ear. This treatment will, however, have no noticeable effect on the mucoid discharge associated with allergy and tubal blockage, except to prevent secondary infection.

A final word: All debris and moisture must be eliminated in order for the dry treatment of infected mastoid cavities to be effective.

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Instruments for Transmeatal Middle Ear Surgery

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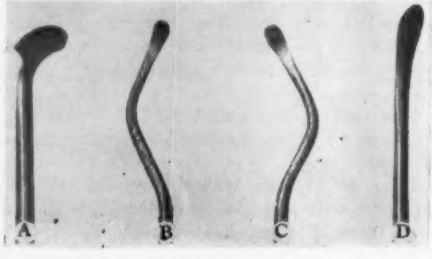
Transmeatal microsurgical interventions in the middle ear (particularly stapes surgery) require, besides the use of optical means (lupes and microscopes) the application of special instruments which allow the necessary manipulations in the narrow visual and operative field. Therefore, we have indicated and we have been using for the past three years an instrumentarium in comparison to other instruments which offers certain advantages. First, offset or bayonet-shaped universal handle; second, instrument tips which can be exchanged and turned; third, malleable instrument tips.

The offset or bayonet universal handle allows the surgeon to operate without visual obstruction.

The ability to turn and to exchange the instruments reduces the necessary numbers; for instance, the same instrument can form a hook or a curette for working above or below or for the right or the left side. Also, the set can be used for the right and left ear and by a right- or a left-handed surgeon.

Finally, the flexibility of the instrument tips allows a maximum distance of the handle from the operative field. Thus, it is possible to reach regions of the middle ear (especially in a trans-

Fig. 1.—*A*, angled knife; *B* and *C*, small angulated dissectors; *D*, large dissector; × 1.5.



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From the Otolaryngological Clinic of the University of Vienna (Head, Prof. Dr. E. Schlander).

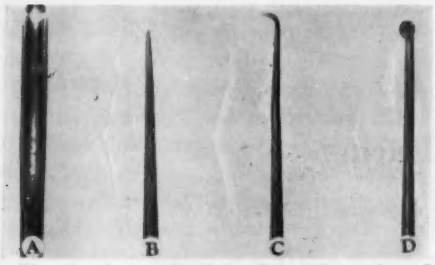

Fig. 2.—*A*, round chisel; *B*, needle probe; *C*, needle hook; *D*, spoon or curette; × 1.5.

meatal approach) which never can be reached with a rigid instrument. The amount of flexibility should only be sufficient to bend the instrument tip during the operation, with enough stability to prevent further bending by the operative procedure. The pointed end naturally is hardened and sharpened as necessary, the same as with any other instruments.

A detailed description and handling of the instruments has been published in the *Monatsschrift für Ohrenheilkunde und Laryngo-Rhinologie*, Vienna.

In detail the instrumentarium consists of the following instruments: universal handles (Fig. 5); one angled knife for the incision of the skin of the external auditorial canal (Fig. 1*A*), large dissector to separate the skin of the external auditorial canal from the bone (Fig. 1*D*), small angulated dissectors to separate the annulus fibrosus of the tympanic membrane from the bony edge (Fig. 1*B* and *C*), chisels of different shapes (round, straight, T-shaped) for removing the posterosuperior bony canal wall (round chisel, Fig. 2*A*), needle probe (Fig. 2*B*), needle hook (Fig. 2*C*), spoon or curette (Fig. 2*D*), mobilizer (Fig. 3*A*), instrument for transincudal mobilization (Fig. 3*B*), curved knives (Fig. 3*C* and *D*), straight chisel for the stapes footplate (Fig. 4*A*), concave chisel for the stapes footplate (Fig. 4*B*), T- and X-shaped chisel for removing the otosclerotic tissue (Fig. 4*C* and *D*). All of these instrument tips are malleable (Fig. 5).

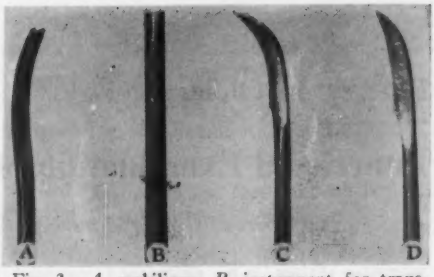

Fig. 3.—*A*, mobilizer; *B*, instrument for transincudal mobilization; *C* and *D*, curved knives; × 1.5.

The above-mentioned instrumentarium is to be used primarily for stapes surgery but can without difficulty be enlarged by the addition of various shapes of tips (curettes, knives, chisels, spoons, hooks, etc.) and can individually be shaped to conform with the requirements of the individual surgeon.

The suction which plays an important role in any kind of microsurgery of the ear is carried out by the use of different sizes of tubes, all of which have the same length and the same flexibility and can be inserted in the same universal handle.

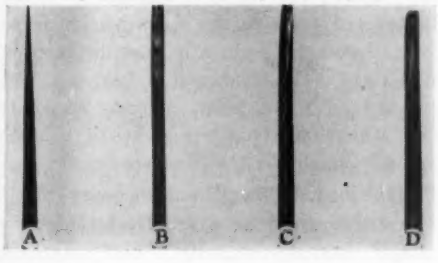

Fig. 4.—*A*, straight chisel; *B*, concave chisel; *C*, T-shaped chisel; *D*, X-shaped chisel; × 1.5.

Summary

The above is a description of an instrumentarium for microsurgery of the middle ear which is characterized by bayonet-shaped arrangements, exchangeable components, and flexibility of the instrument tips.

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Fig. 5.—All of the instrument tips shown are malleable.

Progress in Otolaryngology

Functional Examination of Hearing

Summaries of the Bibliographic Material Available 1957-1958

NORMAN LESHIN, M.D.; SHERMAN L. SHAPIRO, M.D., and LEO A. SATZ, M.D., Chicago

The material in this article represents excerpts from the literature from October, 1957, to December, 1958.* There are also a few abstracts of articles in the foreign literature previous to this time which were not available for the review last year.

Clinical Tests

Rubin [1] emphasizes the importance of utilizing basic tuning-fork tests, which include the Weber, Schwabach, and Rinne tests. An understanding of these tests, the simple pure-tone and speech tests, and accurate observation will enable the average otolaryngologist to evaluate adequately the majority of hearing problems without outside help. This can be easily accomplished with tuning forks and any commercial audiometer. The PB words score, or discrimination score, is the most important test, with word lists which give a most accurate estimation of the patient's understanding ability. This score can be used as a check of the accuracy of the bone-conduction score. Testing for speech is of the utmost importance when surgical procedures are contemplated. The speech-reception threshold is especially of use when checking the accuracy of the pure-tone air-conduction curves. The loss for speech should equal the average loss at frequencies 500, 1,000, and 2,000 in the air-conduction curve. In the category of speech tests, the telephone with special equipment can be used for increasing the fidelity and intensity of the voice. This instrument, the hard-of-hearing telephone, is available as special equipment from the phone company. With an intercom system, it can be used in the office, and, by changing the volume, one can evaluate the ability of the patient to understand speech.

Huizing and Kruisinga [2] report on the result of their investigations of speech audiometry with the use of whispered instead of conversational voice. In constructing intelligibility curves, it was found that in normal persons the whispered-voice curves show a 10 db. displacement to the left relative to that of the speech voice. Owing to the fact that the lower frequencies, which are relatively unimportant in understanding speech, are, to a large extent, absent in whispered-voice tests, the latter can be understood at a lower intensity than spoken material in normal persons. In cases with a rapid fall in the perception of high tones, as indicated by the pure-tone audiogram, the above relationship of the whispered voice to the spoken voice is reversed and the former is now displaced to the right. This makes it a valuable diagnostic tool. The small dynamic range of the whispered-voice test makes it valuable in training children with severe hearing loss.

Huizinga [3] shows the inadequacy of the whispered voice as a reliable test for the social hearing capacity. He refers to the investigation of Huizing and Kruisinga. The strong low components are lacking in the whispered voice. There is a great difference in the change of quality of the spoken voice as compared to the whispered

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* Dr. Alfred Lewy, who was associated with this work for many years, died in December.

voice when speaking into the microphone. In spoken voice, the essential higher components are blurred out by the strong low ones, making the speech more and more distorted and hard to understand. The whispered voice, however, remains clear, with a high grade of intelligibility, though getting weaker and weaker. This can be utilized for the acquisition of speech and language by young children who have a hearing handicap characterized by a sloping audiogram at any grade of hearing loss. In these cases, the child is more easily accessible by means of whispered voice if it is applied in the right way.

Coen and Ghirlanda [4] experimented with the use of the female voice for vocal audiometry, bearing in mind the fact that female speech is usually about one octave higher than corresponding male speech. They employed records made by two suitable voices, one male and one female, using statistically balanced lists of words relayed through the audiometer circuit. A number of cases, including conductive, perception, and mixed forms of deafness, were examined, and the results obtained from each type of voice were compared. Their results indicated that (1) in conductive deafness there was no marked difference in articulation curves whether male or female speech was used; (2) in perception deafness there is a significant divergence, directly proportionate to the high-tone involvement indicated by the pure-tone audiogram; (3) where recruitment exists, female speech gives a more exact result in that the "ceiling" may be more clearly defined than with the usual male record, and (4) in mixed deafness, particularly where recruitment exists, a more exact picture of the index of social capacity may be obtained. The authors consider tests with the female voice essential to the latter criterion in all cases of deafness. They consider, also, that an exact social articulation curve probably should be expressed as an intermediate value between the results obtained from both male and female vocal audiometry. Finally, it is their opinion that female speech audiometry is indispensable for a judgment as to the value of a hearing aid in a given case from the standpoint of the reaction of the cochlea to amplification, transmission of female speech, and evidence of distortion.

Lafon [5] discusses the factor of distortion of phonemes in speech and remarks that the usual lists of words employed for speech audiometry in France are faulty, since they are based upon statistics of frequency of phonemes in written, and not in spoken, language. A proper speech test should include a survey of the auditory field according to the intensity, the various heights, the length of time, and the tonal transitions of the phonemes; tests should be based upon the spoken-voice frequency of sounds, a survey of the badly perceived phonemes, and the manner in which these phonemes are reproduced by the patient.

Qualitative speech audiometry approximating to these standards rests upon the existence in each word of characteristic phonemes required for the understanding of speech. A clinical examination by means of a phonetic test enables us to note distortions depending on the auditory field of the patient. In practice, distortion is most often found in the u, é, o, and â sounds. The i often replaces the e and u. In consonantal phonemes, the j, v, p, and s, are most often ill perceived. The author considers that experimental results and physical analysis of sounds, properly performed, can be compared with a clinical examination of hearing conducted in the customary manner.

In another contribution on this method, Greiner and Lafon [6] discuss the application of the phonetic test to the diagnosis of cortical deafness. This form of hearing deficit is admittedly difficult to recognize. Various tests, including certain modifications of the difference-limen method, the use of filtered frequencies, and tests of perception in noisy surroundings, have been proposed but generally present certain drawbacks which make them impractical.

The authors have applied the phonetically grouped lists of words which they use for vocal audiometry in the otolaryngology clinic at Strasbourg in such a manner as to aid in differentiating cases with language difficulties but no hearing trouble from those with bad phonetic integration due to auditory defects. Furthermore, by the use of these word lists in a proper manner, they have been able to diagnose cortical hearing impairment, even in those instances where pure-tone deafness could not be demonstrated. The list of words containing the appropriate phonemes is read and repeated by the patient at three levels of intensity: 20 db. above the normal threshold of discrimination, 35 db. above, and 50 db. above this level. In clinical practice, it has been found that phonetic distortion is characteristic of perception deafness. If the distortion disappears as the intensity is raised, it is a "liminar" distortion; if it persists despite increasing intensity, it is a "spatial" distortion. There are different types of spatial distortion, but, in general, the authors have found that in cortical lesions after a mild improvement (with elevation of intensity) the distortion (of phonemes) increases with further increase in intensity; on the other hand, distortions due to involvement of the ear itself show an index of distortion at high intensities identical with that shown at moderate levels. The authors express the hope that their small series of cases will be supplemented by tests with their word lists and methods in other hearing centers.

Merklein and Fox [7] report the results of a screening test with the single frequency of 4,000 cps. They used an instrument called the "Oto-Chek (Model 700)" on a group of workmen, presenting the sound at 15 db. above 0 level or, if there was no response, at 35 db. above 0. Tests were made in the personnel office where the noise level varied between 60 and 65 db. After the single frequency test, the men were given the usual threshold test on each ear, with the use of the frequencies in this order: 1,000, 1,500, 2,000, 3,000, 4,000, 6,000, 8,000, 500, and 250 cps. Their investigations confirm previous reports that the 4,000 cps single-frequency test is an accurate test for establishing the upper limit of hearing losses at frequencies below 4,000 cps. They emphasize that they do not consider this test a replacement for the complete discrete-frequency preemployment examination, which should be mandatory for every industrial employee.

Bocca [8] presents a new test for monaural simulation of deafness. This is based on the fact that intelligibility of recorded speech, which is interrupted by equal periods of silence, varies with the number of interruptions per second. On the basis of previous work by himself and others, it can be stated that intelligibility mounts rapidly from 0 to 80%-100% (assuming that the material is delivered at the usual speed of 150 words a minute) as the number of interruptions mounts from 2 to 10 or more per second; intelligibility depends upon the fraction or entire syllable suppressed by the period of silence. It is also known that intelligibility of material, delivered alternately to each ear, is approximately 100%. Taking advantage of these facts, Calearo, of Bocca's clinic, devised a simple test for malingering, which uses recorded material delivered through a commercial audiometer to which is attached a device which alternates the material between the two ears in equal time intervals at a rate which cannot exceed 12-14 "interruptions." The test is begun at a level considerably above threshold (50 db.) and the patient is led to believe that his "good" ear is to be tested. The rate of "alternation" is set at 2 per second—a rate which in testing one ear (periods of speech and silence) would give 0 intelligibility. If the subject repeats at least 9 out of 10 phrases, the intensity level is gradually lowered to 5-10 db. above that of the threshold of the "good" ear for the frequencies 500-2,000 cps. If the subject continues to repeat the material, one can be certain that he is malingering deafness for

speech for the other ear, since if the latter were deaf, the intelligibility curve would behave exactly as it does in testing monaurally, with interruptions of 2 per second, i. e., the patient would be unable to repeat the material accurately.

Harris [9] describes a rapid and simple technique for the detection of nonorganic hearing loss which requires no special accessories beyond the simple basic clinical audiometer. The test utilizes the principle of detecting auditory thresholds by the use of ascending intensities at 10 db. levels, starting at 10 below 0 and then decreasing in 5 db. steps until the subject again fails to respond. Once again, the intensity is increased in 5 db. steps and a threshold established. This procedure is then repeated, by the use of a descending series of presentations in 10 db. steps, starting at 90 db., until the subject fails to respond. At this point, the intensity is increased in 5 db. steps until the subject fails to respond. These thresholds are determined at 500, 1,000, and 2,000 cps. The malingerers utilized in this investigation produced results significantly different from either the normal group or the group with true organic hearing losses. Results showing the descending threshold to be higher than the ascending threshold should be considered suspicious if the difference exceeds 5 db. A difference of 10 db. or greater between the two thresholds is highly indicative of nonorganic involvement. Differences of 5 db. or less cannot be considered as proof of nonmalignancy. This procedure can be given as a screening test, and, where indicated, the battery of tests for nonorganic loss can be administered to the patient before a positive diagnosis is made.

Reilly [10] advocates the use of frequency and amplitude-modulation audiometry for testing the hearing of infants and children. The instrument used is similar to the one Huizing uses in his audiology clinic in the Netherlands. The instrument differs from a pure-tone audiometer by producing a sound which "warbles" to and fro above and below the chosen frequency. The rate of modulation can also be varied. By combining the two methods of varying the sound, a great variety of "warbles" can be produced. The range of the instrument coincides with that of the pure-tone conventional audiometer. The intensity can be varied smoothly from 0 to 100 db., and the intensity-control dial is calibrated in 5 db. steps. For testing the youngest age group, ranging from 6 months to 21 months, the child is held on the mother's lap and portable speakers are placed close to the left and right of the child. The "warbling" sound is introduced from one speaker at different frequencies, starting at 20 db. and increasing by 20 db. if no response is obtained. The usual responses, as movement of the eyes or head, are carefully observed. For slightly older children, up to 33 months, the child is placed at a table playing with toys or blocks. The portable speakers are placed fairly close to the right and left and the sounds introduced. Reception of the sound is usually indicated by turning the head toward the sound. This set-up gives one an approximation of the child's hearing. For older children, warble-tone audiograms are prepared by means of play audiometry, with the use of warble introduced through the speakers at a fixed distance. Later, ear phones are used, and in this way the child becomes acclimated to the procedure so that pure-tone audiograms can be obtained. With the knowledge of sound and testing technique obtained from free-field testing, the child will tolerate the head phones more easily and at a younger age. Pure-tone audiograms can then be obtained at a much younger age than heretofore. Why children respond to a warbling tone at a lower intensity than to a pure tone of the same set frequency is not known. If this proves to be the case, we may have to revise our ideas of threshold-reception levels. A comparison of threshold audiograms for warble and for pure tone in cases of peripheral deafness, central deafness, and cortical auditory disorders would be interesting.

Farrior [11] explains recruitment in a simplified manner and describes some simple tests he employs to determine its presence. He uses the standard audiometer with a microphone and a binaural headset. The binaural headset permits the shifting of the sound from one ear to the other and the masking of one ear while the sound is introduced into the other. The letters A B C are repeated, first in one ear and then in the other. The patient is asked to describe which is loudest and clearest. Beginning at threshold, this is repeated in 10 to 20 db. steps to the 100 db. level. The patient reports when the sound becomes uncomfortably loud and intolerably loud. The results are recorded also by the use of the letters A B C, first as small letters a b c, gradually becoming larger as the sound becomes louder, and then substituting X X for the letter when uncomfortably loud or indistinguishable. The unilateral test he calls the X Y Z, in which one ear is masked and the other tested with increasing intensity. The letters X Y Z are recorded on the ordinary audiogram form. When the otolaryngologist does these brief tests himself, he has the advantage of observing the patient and exercising his clinical judgment as to the presence or absence of recruitment.

Recruitment measured by automatic audiometry was investigated by Landes.[12] Ten normal ears and twelve recruiting ears were given automatic audiometric tests at seven discrete frequencies for comparison with similar data obtained through more standard means. He found that recruiting ears produced narrower threshold excursions at high frequencies than did normal ears under conditions of standard automatic audiometry. With the use of a loudness attenuator in automatic audiometry, a sensitive test of recruitment is provided which uses the criterion of a narrowing of threshold excursions from middle to high frequencies. In automatic audiometry, subjects respond to growth of loudness rather than to difference limens.

Chandler [13] describes a simple and reliable tuning-fork test for recruitment. Although this is a crude test and only qualitative, it is of value because of its simplicity in the clinical evaluation of the unilaterally deafened patient. Any one or all three of the tuning forks of 512, 1,024, and 2,048 cps can be used. The fork is stimulated so that the affected ear can just hear it. The normal ear hears this much louder. The fork is then stimulated forcibly by striking it against the heel of the shoe or some other hard object and immediately presented to each ear in quick succession. The patient who recruits will report equal or near equal loudness in each ear and may even report the sound louder in the affected ear. This procedure is then repeated with the use of the other two forks. A common observation has been a discomfort in the affected ear when presented with a maximally activated fork in the ear with recruitment. The test requires less than one minute of time. This test is not offered as a substitute for tests in wide use now and should be confirmed by more accurate and elaborate tests.

Eisenberg [14] discusses loudness, recruitment, and their value in differential diagnosis. The significant features of the important loudness-balance and difference-limen tests are presented. The conflicting results of present "recruitment" measures lead to the conclusion that no single test constitutes a reliable index for differential diagnosis. The confusion which continues to exist in the field of loudness measurement remains to be resolved by systematic examination of this function in normal as well as in pathological ears, critical evaluation of the test methods now in general clinical use, exploration of new avenues of approach, and increased investigation of the physiological and biochemical processes underlying the growth of auditory sensation.

Hormia [15] studied the difference limen in hearing impairment caused by craniocerebral injury in a series of 43 war veterans. Only four cases showed a partly reduced difference limen. The reduced difference

limen is not characteristic for the hearing impairment in connection with craniocerebral injuries. This does not agree with the prevailing opinion that a reduced difference limen and recruitment are typical of labyrinth deafness. This may be due to the fact that in labyrinthine concussion the inner hair cells are more prone to damage from the concussion wave reaching the labyrinth through the bones. This results in a more uniform damage of the organ of Corti than in those types of end-organ deafness characterized by a reduced difference limen and recruitment.

Pirodda and Segatta [16] conducted an investigation to determine whether the ability to discriminate between variations in intensity in a given frequency was related to tolerance of excessive sound. They used 10 young persons with normal hearing for subjects and employed the Lüscher-Zwislocki difference-limen method. One type of audiometer was used, but two other makes were used for controls. A comparison was made between the results of this test and the results obtained by overloading the ear in the same persons with a pure-tone stimulus of 2,000 cps at 100 db. intensity for two and four minutes. It was found, as the literature already indicates, that there are variations within certain amplitude in the values obtained in normal subjects with the Lüscher-Zwislocki test. It was also found that low values of difference limen (DL) correspond to a very remarkable fatigue effect (as expressed by the amount of the threshold elevation for a test tone of 3,000 cps as well as the time required for the restoration of the prestimulatory threshold), while this fatigue was very slight in those subjects showing high values of the DL. An intermediate behavior was observed in subjects with intermediate values of the DL. The relationship between the two tests suggests that the determination of the DL may take the place of the so-called fatigue test as the easier and less time-consuming procedure in determining susceptibility to noise.

Jerger and associates [17] report their clinical observations on excessive threshold adaptation in two cases of acoustic neurinoma. They found that these patients required more and more intensity to keep the tone at threshold under continuous stimulation. This is brought out by first testing with the interrupted stimulus and then with the fixed continuous frequency. The former remains relatively stable, whereas the continuous tones show the patient requiring considerably more intensity to maintain the stable hearing level. This becomes evident for frequencies above 170 cps and becomes definitely evident by the time the frequency reaches 250 cps. These findings are exactly opposite to the earlier observations of Dix and Hood, who reported that threshold adaptation exemplified by the gradual decline in the threshold-tracing overtime characterizes certain features of cochlear as opposed to retrocochlear, eighth nerve pathology. It is urged that threshold tracings for continuous tones of fixed-frequency overtime be obtained in all cases of suspected eighth nerve lesion, as this may be one of the few, if not the only, auditory symptom that can be used reliably to differentiate cochlear from eighth nerve lesion.

König [18] investigated the effects of loudness and age on the differential sensitivity to frequency. The effects of age on the size of the difference limen for frequency were determined at a sensational level of 40 db. above threshold, the reference frequencies being 125 to 4,000 cps. It was found that the ability to detect small changes in pitch apparently begins to be affected at all the audible-frequency ranges as early as the fourth decade of life. Between the ages of 25 and 55 years, the pitch-discrimination performances seem to deteriorate in approximately linear manner. After 55 years, the size of the difference limen increases more abruptly. At the seventh decade of life, the relative deterioration of the differential thresholds are about 300% at the higher and lower frequency ranges and 200% at the middle frequencies. The in-

crease in the size of the difference limen for frequency with the advancing age appears to have some connection with the degeneration of some nerve fibers which normally participate in the transmission to the brain of the different tones.

Harris and co-workers [19] made a test of the important thought of Miskolczy-Fodor that the way a partially deafened ear integrates energy over a duration of time may provide a clue to the location of the disorder. They were unable to confirm the opinion that integration data will in all patients allow one to separate conductive and perceptive components, or that atypical integration data always and only accompanied recruitment. They found some types of intracochlear disorder exhibiting both; some types, neither, and some types, either one alone. However, they feel that the Miskolczy-Fodor test can have a very real place in a completely equipped diagnostic clinic, and they trust that mounting data from their laboratory and others will eventuate in a clear picture.

Miller and Fowler [20] describe a new method for performing speech audiometry, with the use of Magnetic Cards on tape. The instrument, the "Language Master," is an electronic device based on the principle of the playback of a tape recorder. It uses the magnetic tape applied to a card and can be used for speech audiometry. A single spondee, or phonetically balanced word, is recorded on each card along with a carrier phrase. Each test set contains a card on which is recorded a 1,000-cycle tone for calibration purposes. Prior to the test, the cards are scrambled so that a variety of word orders are possible with a given test. The same list can be used several times during a test without the memory factor affecting the results obtained. The examiner can also communicate with the patient through the microphone during the course of the evaluation, while the actual test material is presented via the specially prepared cards. The instrument appears to permit the flexibility of microphone voice testing and the reliability of recorded voice testing. It is suggested that different institutions explore the use of the instrument in their diagnostic and research problems.

Myers [21] notes that the assumption regarding the use of discrete frequencies as customarily practiced in audiometry for the purpose of gauging the integrity of the intermediate frequencies is not always correct. The Békésy type audiometers, while giving a more accurate picture of the state of the intermediate frequencies, are expensive and time-consuming. For this reason, the author experimented with the use of bands of noise as substitutes for pure tones in testing hearing. A group of 12 patients with partially defective hearing was examined with tones of 500, 1,000, 2,000, and 4,000 cps and also with bands of noise centered at those same frequencies. No important advantage in test reliability was observed for either type of stimulus. The correlation between the two measures decreased slightly with higher frequencies. Thus, it is suggested that if a Békésy-type audiometer is not available to examine patients with high-tone losses, testing with a photograph disk containing one or more narrow bands of noise might add more information to that yielded by discrete-frequency audiometry.

Experiments and investigation by Zarcoff [22] show that it is possible to predict the speech-reception threshold of a person from a white-noise threshold with precision entirely adequate for clinical purposes. Apparently, the spread of acoustical energy along the basilar membrane is accomplished in a similar manner by both speech and noise, in effect averaging the different frequency contributions to the signal. The continuous quality of complex sounds in contrast to the discrete quality of pure tones would lead one to expect these relationships. Substituting the white noise for the Speech Reception Test permits one to avoid such problems as language difficulties, learning, and excessive expenditures of time. A suitable stimulus is any broad-band noise, such as is available in many hearing clinics. The

high correlation between the white-noise threshold and the speech-reception threshold is related to Fletcher's formula of the best two averages between 500 and 2,000 cycles.

Hood [23] discusses the principles of bone-conduction audiometry. He agrees with Denes and Naunton that a masking noise should have "good masking ability for minimal loudness." White noise, which has to be at least 20 db. higher in intensity than the pure tone which is being masked, attains a considerable increase in loudness at that level and is not ideal for masking the opposite ear in bone-conduction tests. He favors the use of narrow, so-called critical, bands around the frequency which is being tested. For this purpose, he employs a white-noise generator in which a number of filters of high sensitivity are centered around each of the pure tones normally used in bone-conduction audiometry; it is also hooked up to an attenuator calibrated in steps of 10 db. This device will give an exact 1:1 relationship between noise level and masked threshold. The complete procedure is as follows: 1. Make an air-conduction audiogram of both ears in the normal way with masking, if necessary, of the untested ear when the difference in hearing loss between the two ears exceeds 50 db., which is attenuation level for air conduction. 2. Find the bone conduction with the conductor at the mastoid of the tested ear without the use of masking. 3. Apply the masking noise of the appropriate band of the untested ear by means of a special insert receiver in order to find the bone-conduction-threshold reading. 4. Use "shadowing procedure" by employing increasing intensity of masking noise in 10 db. steps until that point is reached at which the bone conduction remains constant regardless of further increases of masking noise. This is a clear discussion of a debatable subject with good explanatory diagrams.

In explaining the complicated mechanism that takes place in the lateralization of the Weber test, Langenbeck [24] expresses the opinion that in the Weber experiment there is not a question of the bone-conduction tone in one ear being perceived louder than in the other but that we are dealing rather with a two-directional type of hearing. The bone-conduction tone arising from the top of the head, or from the middle of the forehead, arrives temporally much sooner in the inner ear of the sound conduction—defective site—than in the inner ear of the sound conduction—normal site. The complicated lateralization process becomes more understandable when we realize that in stimulating bone conduction two kinds of physical developments come into being: (1) air-sound waves in the external auditory meatus and (2) periodic pressures and subpressures in the labyrinthine fluid, according to the rhythm of the bone-conduction vibrations. Both together effect fluid current vibrations in the inner ear, which lead to excitement of the organ of Corti and thereby to the production of the bone-conduction tone.

After explaining in detail the different types of vibrations that occur in the skull during the process of production of bone conduction and its propagation to the labyrinthine fluids, he concludes that the Weber lateralization phenomenon rests upon phase differences in the stimulation of the two inner ears. It also becomes clear that the bone-conduction tone arrives in the sound condition—defective ear—a moment sooner to perception than in the sound conduction—normal ear.

A tuning fork placed on top of the skull of normal test subjects outside the midline may be heard, depending on place and frequency, in the contralateral ear. This phenomenon was investigated by placing a bone-conduction receiver along an adhesive strip (attached to the head of the observer) running from one ear over the top of the skull to the other ear. In these investigations, Spoor, Schmidt and Van Dishoeck [25] established the following facts: For frequencies around 500 cps there was one region on each side of the skull where placing of the receiver gives lateralization of

the tone to the contralateral ear. For frequencies around 1,200 cps there is on each side of the skull a region with lateralization to the ipsilateral side and one with lateralization to the contralateral side. The writers think that these lateralization impressions are due to interaural intensity relations rather than to interaural phase differences. These intensity differences are due to regions of minimal and maximal amplitude existing in the vibrating skull.

In this paper Groen and Hoogland [26] analyze a case of otosclerosis of the round window from the point of view of bone conduction. First, they attempt to explain the mechanical and physiological forces underlying the production of normal bone conduction; the inter-relationship between the vibrations of the skull produced in bone-conduction testing; the formation of pressure waves along the bony enclosure of the cochlea, and the displacement of the labyrinthine fluid in the two scalae and its effect on the cochlear duct, which is thought to be responsible for the production of the bone-conduction tone. They also explain the pathway of the acoustic leak that takes place in this process of normal bone conduction as well as of the acoustic leak at the scala tympani along the apertures of the cochlear aqueduct and the inferior cochlear vein that takes place in the production of bone conduction in otosclerosis of the round window. This latter fact is considered to be responsible for the bone-conduction audiogram in otosclerosis of the round window before the operation. The practical results are demonstrated in a case of a complete occlusion of the round window niche.

Harbert [27] recommends the routine use of 100 db. re 0.0002 dynes per square centimeter of white noise for masking in both air- and bone-conduction measurements. The white (random) noise is a mixture of all frequencies in approximately equal proportions. A white noise delivered by standard receivers to a normal ear in a reasonably quiet room has to attain a level of 50 db. re 0.0002 dynes per square centimeter before any effect is observed on crossed hearing as measured by bone conduction at the opposite ear. Crossed masking occurs under the same conditions whenever the intensity is raised by an amount equal to the attenuation of the skull plus the ineffective component of the mask. This level for white noise is 100 db. re 0.0002 dynes per square centimeter. The shortcomings of commercially available masking sounds to eliminate a conductively deafened ear are pointed out. Most machines are calibrated on a purely arbitrary basis, and dial settings differ widely with different manufacturers. The spectrum also is not specified. He suggests that a uniform standard of masking be prescribed by some standards committee or organization and that a standard measurable reference and a dial calibrated in decibels above this level would end this confusion. More effective methods for masking should be developed, such as the use of narrow bands of noise to reduce the ineffective portion of the total stimulus or utilization of the Stenger principle, where the same sound for masking and stimulus is used but varying one by intensity modulation to permit differentiation.

Especially in cases of conductive deafness, one may encounter great difficulties in testing each ear separately by bone conduction. To attain more reliable results when testing bone conduction, one must pay special attention to the methods of masking used. Palva [28] feels that the clinical use of masking would be greatly facilitated if, with a convenient device and white noise with uniform spectrum, the interaural attenuation could be raised to a higer decibel level. The author has, therefore, experimented with two types of meatus inserts suitable for use in bone-conduction testing of the ears to see whether or not the interaural attenuation could be raised to make such methods worth while.

Dieroff [29] attempts to find a reliable and portable masking device. Since the Bárány noise apparatus has been in use in many

clinics in such capacity, the author investigated 11 Bárány noise apparatus found at his institution to determine their scientific reliability as masking contraptions. He reports his observations regarding sound-pressure measurements and frequency analysis on the 11 Bárány drums. He used an artificial ear, a Terz filter, and an automatic writing machine for his investigations. His investigations confirm the long-known fact that this manner of masking with the Bárány drum is unsuitable scientifically, since the ear to be examined is uncontrollably overmasked.

Lüscher and Kusen [30] state that, according to their investigations, the reflex audiometry permits determination of threshold of hearing without subjective information from the patient. Thus, reflex audiometry can replace ordinary audiometry in cases where, for some reason or another, the latter cannot be performed. Reflex audiometry is successful only in a certain percentage of patients. One can make use of this method only if we deal with incontestible and repeatedly confirmed curves with doubtless successful conditioning and tone reaction within known reaction times.

No conclusion may be drawn from negative results; nor may presence of hearing be excluded by negative results. The ascertained threshold of hearing lies, with few exceptions, below the subjective threshold values, and one may reckon with up to 20 db. and in exceptional cases up to 30 db.

Charan and Goldstein,[31] of the Central Institute for the Deaf in St. Louis, discuss the variations and anomalies in the psychogalvanic test for hearing. They state that, while the use of this test is gaining ground, there are a number of references in the literature on the difficulty of eliciting electrodermal responses (EDR) in some patients. In the authors' experience, it was more difficult to condition electrodermal responses in children whose auditory disorder may have reflected some defect of the central nervous system than to condition EDR in children whose hearing loss probably resulted from a defect of the cochlea or auditory nerve; difficulty was also found in conditioning some adults suspected of having a nonorganic hearing loss. However, the difficulty of conditioning depressed electrodermal responsiveness in general could not simply be attributed to some physiologic or psychologic dysfunction. Observations on 32 normal children indicated that nearly half of them were difficult to condition with the techniques used, and similar results were obtained in exploratory work with some normal adults. They conducted experiments on 16 men and 12 women between the ages of 17 and 41, all with normal hearing except 1, for the purpose of exploring the relationship between the EEG pattern and the ease of eliciting electrodermal responses. They concluded that (1) there are normal adults in whom it is difficult to elicit conditioned electrodermal responses to sound and (2) the presence of a strong α-pattern in a male may be inversely related to the ease of eliciting conditioned electrodermal responses; this relation does not hold true for females. The findings apply to normal adults; they may or may not apply to children.

Lehrhoff [32] made an experimental study of the relationship between auditory thresholds in normal children with normal hearing and in cerebral palsied children with normal hearing by the use of pure-tone air conduction and the psychogalvanic skin response. He found a close relationship between the auditory thresholds of these two groups for the two methods of procedure. The psychogalvanic skin response (PGSR)-threshold means were 4 db. below those obtained by the pure-tone air-conduction methods. The galvanic skin response did not yield reliable and valid results with the cerebral palsied children. It was radically different from that of the normal. This may be due to the fact that the response is at least in part cortically controlled. The latent periods of this response differed significantly at the 1% level for the normal and the cerebral-palsied groups. Differences be-

tween frequencies for the two groups were only chance variations. There was no significant difference between the right and left ears for either group. PGSR audiometry should be used with caution with all cerebral-palsied children until further research is completed. It is a test to be used as an aid to other testing procedures and not as an ultimate end.

Strauss [33] reports on the effects of mephenesin carbamate on normal hearing thresholds as determined by the conditioned psychogalvanic skin response (PGSR) and conventional pure-tone audiometry. Active-going children and hyperactive patients with neuromotor disorders cannot be tested satisfactorily by PGSR audiometry because their activity interferes with the correct interpretation of the galvanic responses. Something to reduce limb and body motor activity without affecting hearing thresholds is needed. Mephenesin carbamate does this. A group of normally hearing persons were tested after being given the drug. Tests showed that this drug has no statistically significant effect upon hearing thresholds as determined by PGSR audiometry and conventional pure-tone audiometry. It is suggested that this drug be used as premedication in clinical audiometry in order to facilitate the testing of those patients whose hearing thresholds cannot be evaluated satisfactorily by existing methods because of interfering motor activity.

Sanchez Longo, Forster, and Auth [34] discuss the literature on sound localization (which is extensive), as well as the anatomic pathways involved, and present a clinical test to determine a given person's ability to localize a source of sound coming from a horizontal plane. In order to do this, they conducted experiments on normal persons who were blindfolded and placed on a stool facing a Ferree-Rand type of perimeter with an arc of 330 mm. radius; the head was fixed to eliminate vestibular and head-movement sensations. The examiner carries a pure-tone audiometer received along the arc of the perimeter, which emits a complex sound from the masking selector at 90 db. intensity; this sound has a fundamental frequency of 60 cps and is delivered from 13 different positions at 15 degrees from each other on the left and right sides. The patient holds a metal pointer in each hand, with which he points toward the source of the sound. In addition to the normal subjects who showed an average angular deflection of 7.2 degrees, five cases of temporal bone lesions were studied, all of which showed impairment of accurate sound localization in the contralateral auditory field. The authors believe that sound localization in the horizontal meridian is a specific biologic function which is altered by unilateral temporal lobe damage, but the specific area in this lobe has not yet been defined. For localization of sound from above or below or from in front or behind the head, head movements and vestibular cues are required. For the localization of the distance of the source of sound, a limited ability to accomplish this purpose is afforded by the total intensity of the sound. This is a thorough review of existing information on the physiology and anatomy of the subject, which should be read in the original for those interested.

Directional hearing was examined in a group of patients suffering from various types of loss of hearing: conductive loss, nerve deafness, otosclerosis, and unilateral total deafness. The investigations of Jongkees and van der Veer [35] reveal that neither conduction deafness nor a difference in hearing acuity of the two ears necessarily results in an impairment of directional hearing. The perception-deafness group often showed impaired directional hearing. Patients with otosclerosis also showed disturbed directional hearing. Unilateral total deafness does not necessarily prevent directional hearing.

It is the authors' impression that in these patients a change in time-and-phase pattern may cause the loss in directional hearing.

Tests were performed by Jongkees and van der Veer [36] on a subject seated within a graduated arc with his eyes closed. A

1,000 cps tone was presented, and the subject was asked to indicate on the arc with his fingers the subjective location of the sound, that is, the direction from which he perceived it. The subject generally indicated this location too far laterally on each side. This experiment shows that there is not a perfect unity between objective and subjective sound location if directional hearing is examined in the horizontal plane. A graph made of the subjective site as function of the objective one always presents a characteristic S-form. This form cannot be influenced by varying the position of the head or the method of indication.

Directional hearing is usually explained by differences in time, phase, and intensity of the sound waves presented to the two ears. This, however, does not well explain the fact that in Jongkees and van der Veer's [37] investigation of 10 patients with unilateral total deafness normal directional hearing was found in 2 patients and fairly normal hearing in 1. In these cases, time, phase, and intensity differences in the two ears could not possibly explain the phenomenon of directional hearing. In unilateral deafness, we think that directional hearing is made possible either (1) by the influence of the vestibular apparatus, (2) by movements of the head, or (3) by the influence of the auricle. The writers' examinations were directed toward investigating the participation of these three factors in the directional hearing.

In conclusion, they learned (1) that they could not find any direct influence of the vestibular organ upon the directional hearing; (2) that in very special circumstances small movements of the head may help in the directional hearing sensation (neck reflexes and vestibular reflexes have some influence on these movements, but their importance is not very great); (3) that the influence of the auricle is more or less of a shielding nature and varies for tones of different pitch (the auricle influence is especially strong in frequencies higher than 5,000 cps.).

Problems in military audiometry were discussed in a symposium [38] given under the auspices of the Armed Forces Research Council Committee on Hearing and Bio-Acoustics. A number of contributors discussed various aspects of hearing tests as applied to military problems. The question of degree of accuracy—considering the large numbers of men requiring testing on certain occasions, as well as the limited availability of testing personnel—the frequency with which hearing tests should be made, the new problem of continuous exposure of personnel to high-intensity noise, the type of hearing tests required to determine whether or not a hearing loss is service-connected, and other problems were taken up in short separate discussions. Some of the leading points brought out included the fact that while the customary test at induction is the classic spoken-voice test, at the time of discharge or shortly thereafter a spoken-voice test plus a pure-tone test may be given. If the latter shows a significant loss, the man is eligible for compensation regardless of the fact that this may have existed at the time of induction in spite of a "normal" spoken-voice test on his record. Pure-tone examinations are therefore recommended at the time of induction and discharge. Other papers dealt with the desirability of amplifying hearing tests at induction and discharge to include additional standard diagnostic methods as well as a classification of hearing tests.

Considerable space was used for discussing automatic audiometry and various types of automatic audiometers as well as appropriate methods for using them in the armed forces, especially for screening purposes. It was concluded that the group screening technique developed by Dr. Glorig was a recommended procedure but was applicable only to induction centers where large numbers of men must be processed; it was also concluded that, at a minimum, a pure-tone audiogram at induction and at discharge should be an indispensable part of the general examination.

Gleason [39] reports an investigation made at the Audiology and Speech Center of Walter Reed Army Hospital, where an attempt was made to determine the psychological characteristics of patients presenting inconsistent audiological-response patterns. Thirty per cent of 278 consecutive military patients revealed inconsistencies during audiological testing which resulted in an exaggeration of their hearing loss. In 86% of the patients, such functional overlay was associated with a medically diagnosed organic hearing loss. Psychological testing and personal interviews of these inconsistent patients showed a poorer intellectual performance, a greater degree of psychiatric and psychosomatic complaints, and deviant social behavior and ethical patterns. These audiological-inconsistent patients present a high failure rate in adjusting to their hearing loss. This group is heterogeneous and composed of three psychological subclasses. The largest group demonstrates emotional immaturity, instability, and inadequate response to social demands. The next largest group is a neurotic population high in anxiety and feelings of inferiority and lacking in self-confidence. The third small group appears to be normal psychologically but needs further study. All experience this exaggeration largely as an unconscious process. There were only a very small number of apparent malingerers.

Traumatic and Occupational Deafness

Scal,[40] in discussing occupational deafness, states that a diagnosis of acoustic trauma can be made only when the causative factors are evident. This implies constant exposure to noise above the 90 db. level for a very long period of time. When there is a question about the noise level at a given place of occupation, it should be determined by a noise-level meter so that workers can be informed of the hazard of the noise. Preemployment audiometer tests, repeated at regular intervals, should be performed on all workers in noisy occupations. Such tests will disclose susceptible subjects. Those with conductive deafness should not be excluded, as the condition acts as a barrier and natural plug against harmful vibrations.

A study was made by Lierle and Reger [41] of the effect of tractor noise on auditory sensitivity. Eighty full-time tractor operators with negative ear histories were tested. Measurements of tractor noise showed that the sound pressure level exceeded the 85 db. critical level, ranging from 88 to 102.5 db. in the various cycle bands. This, they feel, is sufficiently high in intensity to produce high-frequency hearing losses in tractor operators with noise-susceptible ears if exposed over a period of several years. The intensity of this noise would be reduced with the use of more effective mufflers and the utilization of vibration-control construction methods. High-frequency dips with the maximum at 4,000 cycles were found and were commoner between the 30- and 60-year age levels. Slightly greater losses occurred in the left than in the right ears. The fact that dips in elderly persons were not found does not rule out the possibility of some noise-induced hearing loss. Those with noise-susceptible ears experienced tinnitus. These people should wear effective ear plugs. They recommend that hearing surveys in the future be designed to test sensitivity at 500, 1,000, 2,000, 3,000, 4,000, 6,000, and 8,000 cps, so as to obtain more information about the frequency-intensity characteristics of dips and to learn more about the aging ear. This is a preliminary study, and findings should be considered tentative until confirmed by a larger number of auditory measurements.

Hansen and Westerbeck [42] carried out an investigation of hearing in a squadron of pet aircraft mechanics (141 men) to determine hearing losses due to exposure to the noise of the plane engines. Examination included (1) a case history, (2) otorhinologic examination, and (3) two audiograms—the first one performed not less than 4 hours after the last noise exposure and the second one made 90 days later. The Hearing Data Conservation Card originally prepared by the Subcommittee on Noise in Industry, and

later modified by a CHABA working group, was used. The results of the "reference audiogram"—the one taken 40 hours after the last noise exposure—showed that 40% of the men demonstrated no more than a 15 db. loss at any frequency and 55% showed a hearing loss of 20 db. at some test frequency but did not qualify for the next group of 6%, which indicated an average loss of 20 db. or more at 500, 1,000, and 2,000 cps. After three months of routine noise exposure, only three men in the first group showed hearing losses of 20 db. or more in some frequency, while six men showed improvement. In the second group, 11 men showed an increased loss, while 20 men showed improvement. There was nothing in the medical history that would correlate the apparent differences between the three hearing groups, and there was no difference between the first and the second group as to the use of protective devices. The study was made to assist in proposed revision of regulations governing hazardous noise exposure in the air force.

Pestalozza[43] has studied the fatigue effect obtained by continuous and interrupted noise on the 4,000 cps frequency. Duration of stimulus has been of two minutes, and the intensity level reached 95 db. and 105 db. over threshold. Interruption frequencies of between 2 per second and 1,000 per second have been tested. The relationship between noise and silence was kept at 1:1. Results obtained in five subjects showed that at an intensity level of 95 db. a noise interrupted more than 100 times per second gives the same fatigue effect as a continuous noise; on the contrary, at a 105 db. level of intensity variously interrupted noise gives rise to less fatigue than continuous noise, even if there appears to exist a relationship between frequency of interruptions and fatigue effects.

If one bears in mind that the relationship of noise-silence equalling 1:1 brings about a decrease of the mean level of interrupted noise reaching about 6 db., one has observed that the fatigue effect due to a continuous noise at 95 db. is inferior to the one of an interrupted noise at 105 db. (mean value 99 db.). This signifies that the fatigue effect of an interrupted noise of high intensity is due more to its mean level than to its highest peak level (author's abstract).

Barron[44] conducted studies on 470 flight-line mechanics who have been exposed to reciprocating- and turbojet-engine noise for periods up to seven years while employed in an air-frame manufacturing company. In the immediate vicinity of these engines the noise levels may reach peaks up to 150 db., and levels of over 100 db. may be expected under any condition when the engines are running. However, exposure periods were limited to an average of 60-90 minutes, with less than 15 minutes at maximum levels, and physical approach to the engine noise source are limited by the whirling propellers. Barron found that there were no significant changes in the mean tests for the group at 1,000 or 4,000 cps frequencies other than those generally associated with presbyacusis; individual threshold changes revealed only six cases of elevations in excess of 20 db. at 4,000 cps. Threshold changes appeared to be unrelated to the use of ear protection.

Hinchcliffe and Wheeler,[45] in a study made at the Royal Air Force Acoustics Laboratory, measured the ability to receive speech over the telecommunications system by aircrews after a long flight. Previous investigations had shown appreciable elevation of the pure-tone threshold (presumably caused by the constant noise of the engines [abstract]) due to auditory fatigue —a loss which was calculated to be equivalent to hearing losses for speech of 20 db. or more. Inasmuch as the telecommunication system functions against a background of noise, tests were conducted in a similar fashion. A phonetically balanced word list, disk-recorded against a background of aircraft noise, was used; the records were played to reproduce the noise at a sound pressure level of 100 db. and the speech at 106 db. at the ears. It was found that a

long operational flight had practically no deleterious effect on the crew's hearing efficiency for speech, as contrasted with the significant raising of the pure-tone threshold. The authors consider that the difference demonstrated by the two tests is due to the fact that in acoustic trauma or temporary deafness, where the hearing loss is less than 30 db., only the external hair cells of the basilar membrane are affected and the internal hair cells are intact; owing to recruitment, which is characteristic of defects occurring in acoustic trauma, the reception of speech at 40 db. or greater (provided there is no discrimination loss) is therefore not diminished.

Bernabei and Carmi [46] report the results of otologic studies among the workers in the Travertine quarries, where pneumatic hammers are in constant use; noise-level measurements indicated that the intensity often reaches 120 db., with a predominance of the middle and high frequencies. A total of 128 workers were examined as to the appearance of the ears, nose and throat; subjective complaints, and audiometric pure-tone studies. A chart is attached, showing the losses for each frequency in different groups of workers, ranging from those who had been employed for only 5 years to those who had worked for over 25 years in the quarries. The authors point out the frequency and progressiveness of the hearing deficit which is of a perceptive character with an initial dip at 4,000 cps. They believe that not only the intensity of the sound and the frequency characteristics but the vibration imparted to the body by the instrument and individual predisposition are factors in causing the hearing impairment which in no case reached the state of total deafness. They advocate repeated audiometric examinations, reduction of the noise, suitable ear plugs, and elimination of concomitant physical defects which might serve to aggravate the hearing loss as suitable prophylactic measures.

Glorig et al.[47] are conducting a five-year laboratory study of temporary threshold shift (TTS). This was initiated with a pilot study. The TTS is a consistent repeatable phenomenon. There are wide individual variations in the amount of TTS by a given noise exposure. Few have very large or very small shifts, the majority are clustered about the average value. The growth and recovery of the temporary shift at all frequencies tested, 1,000 to 6,000 cps, are logarithmic functions of time of the general form.

$$\text{TTS} = \text{const.} \times \frac{\log \text{exposure (or recovery)}}{\text{time} + \text{const.}}$$

The magnitude of the shift is larger at the higher frequencies. Control data indicate that practice effects did not influence the results. This pilot study shows that the temporary threshold shift is a stable phenomenon and that a long-term program of research into the nature of TTS is entirely feasible.

Further studies of this temporary threshold shift (p. 838) showed that the TTS produced by intermittent exposure to noise is significantly less than the shift produced by continuous exposure to the same total amount of noise. The TTS produced at 4,000 cps by exposure to broad-band noise is significantly larger than the shift at 1,000 cps. At the former, shifts of more than 50 db. recover much slower than do shifts of less than 50 db. The maximum shift produced by exposure to octave bands of noise occurs on the average one-half octave to one octave above the nominal high cut-off frequency of the exposure band. The measurement of TTS may be a possible method of assessing noise exposures and may eventually be used to predict the amount of permanent threshold shift to be expected from extended exposure to that noise.

De Mitri and Pasini [48] investigated the intelligibility of speech when various types of ear protectors of the stopple type are used, both in silent and in noisy surroundings. This touches upon the important problem of preventing damage to the hearing from excessive noise while at the same time making it possible for the workman to hear instructions and other essential speech even

when wearing the ear stopple. Various types of these devices were tested, both nonselective and selective, i. e., those meant to screen out mainly harmful frequencies; some of these are makes which are familiar in the United States. It was found that selective as well as nonselective ear stopples when used in silent surroundings produce a uniform rise of about 10 db. of the thresholds of detectability, perception, and intelligibility of speech. In noisy surroundings, on the other hand, the threshold of perception is lowered, facilitating the understanding of speech. A device known as selectone K, which is a selective ear stopple with a low-pass filter, enabled subjects wearing it to reach 100% of intelligibility of speech while furnishing protection from the damaging effects of high frequencies.

Rehabilitation

A comprehensive listing[49] of the audiology and hearing-rehabilitation centers in the United States is made available to the otolaryngologist so that he may refer his patients for competent hearing testing and educational rehabilitation. It also gives information regarding the status of the personnel of the centers.

Mrs. Tracy[50] makes a plea for the early detection of deafness in infants and for proper preschool education. This includes guidance, training, and education of the parents. The program of the Tracy Clinic is presented, as well as the correspondence course which is available to parents of children between the ages of 2 and 5 years, inclusive.

Williams and Ling[51] discuss the value of speech audiometry in the training of deaf children. The writers state that they could find no record of any controlled experiment in which the hearing for speech of school-age children was assessed by speech audiometry before and after auditory training. It is known that conductive deafness can be overcome and the patient's hearing for speech brought to normal by the use of a hearing aid; perceptive deafness, even if slight as measured by the pure-tone audiogram, may prevent words being heard correctly even if the intensity is greatly amplified. They carried out a training program, after tests for pure tones and speech reception with suitable lists of words, on a group of partially deaf children. The method consisted of normal class lessons and stories recorded at approximately constant levels of intensity as well as a wide variety of speech and other sounds; the group aid was used during the course of the study for an average of 20 hours weekly. All cases initially were intolerant to sound intensity above certain levels. By working just below these and increasing the intensity in 5 db. steps over a period of months, all intolerance up to 115 db. was overcome. It was found at the end of a nine-month period that monaural speech discrimination was raised from an average maximum of 43% to 85% and that the range of intensities over which words could be recognized was considerably extended.

Campanelli et al.[52] made an otoaudiological evaluation of 44 premature children in the age range of 72 to 84 months. A similar control group was also tested. Both groups had essentially negative otolaryngological findings regarding external and middle ear pathology. Audiological measurements were within normal limits for both pure-tone and speech stimuli. The premature children had seven cases (15.9%), with a bilateral sensory-neural hearing impairment from a moderate to a severe degree of loss. These results should alert the clinical worker to suspect the presence of a sensory-neural hearing loss whenever a premature child is examined, particularly where there has been a history of a very low birth weight and a long period of incubation. From the findings reported, no one single agent could be named as being responsible for the auditory defect. Continued investigation is suggested to explore the possibilities inherent in this particular pediatric problem.

Armando[53] reports on the results of screening tests carried out in the province of Parma, in Italy. A total of 2,216 school children were examined. Of these, about 400 were tested with the Massachusetts Hearing Technique (512-1,024-4,096-8,192-11,584) at intensities of 10-15-15-20-25 db., with the use of a Maico F-1 Audiometer with multiple receivers allowing 10 ears to be examined at each sitting. The remainder of the children were examined with the sweep-check method. Those subjects who showed a loss of more than 20 db. were given a more detailed examination, including a study of the ears, nose, and throat and a complete audiometric study in a silent room at the otologic clinic of the University of Parma. A total of 6.09% of the children examined showed a hearing deficit of significant proportions; most of these cases were susceptible to treatment, but 1.85% of them were judged to require special schooling.

Johnsen[54] discusses the results of a survey of 109,400 children attending schools for normal children in Copenhagen. All children in municipal schools are tested annually with whispered voice; those who are unable to hear whispered voice in one or both ears at a distance of at least 6 meters are examined by the author. If the otologic examination does not reveal the cause of the hearing impairment or if whispered voice, at the repeated test, can only be heard at a distance of 3 meters or less, pure-tone audiometry is performed in a soundproof room. Out of the above total of children tested during the period of 1947-1951, two hundred twenty proved to be suffering from hearing impairment of the perceptive type. Four different audiometric patterns were found and a correlation indicated between the etiology of the hearing loss and the audiometric pattern. Inherited perceptive deafness gives chiefly a graph sloping gradually toward the high frequencies, whereas acquired perceptive deafness gives sharply sloping curves. Audiograms representing cases in which the etiology is unclear belong in most cases to the gradually sloping type. It seems justified, therefore, to conclude that a large proportion of these cases are inherited.

Sortini[55] comments on the difficulty of testing preschool children by routine pure-tone audiometric tests and analyzes the results of the psychogalvanic test made on 250 preschool children at the Hearing and Speech Clinic of the Children's Medical Center, in Boston, during a one-year period. Ages ranged up to 5 years, and the children are classified in five groups. Losses of less than 20 db. were considered to be within normal. Of the total number examined, 185 children were found to be hearing-handicapped, with losses grouped into four classes from "mild" to "profound"; not one child was found to be totally deaf, some degree of usable hearing being found in every patient tested. The hearing handicapped children were classified according to audiologic and educative levels. Information is presented concerning the distribution of recommended and fitted hearing aids in the various age levels represented in the group with hearing loss. Of 174 potential hearing-aid users, 155 (89%) were fitted as soon as possible after audiologic work-up and exclusion of medical aid. The probable etiology of the hearing losses is also considered.

Terrier[56] discusses the method used for screening children with defective hearing at Lausanne, Switzerland. Owing to limitations of time and personnel, it was decided to limit the test to children who had reached the age of 10 years. The sweep-check method at 15 db. of all frequencies on the audiometer, leaving out 125 cps and 16,000 cps, was used; those children who failed to hear three frequencies or two speech frequencies (500 to 3,000) were given a complete examination, including a complete audiogram. Of the 637 children examined, 29, or 4%, were found to have defective hearing involving various types of impairment. No therapy was given, but the parents were notified in these instances of the defect and advised to consult an otologist.

Geyer and Yankauer [57] give results of hearing tests on 461 children of preschool age (from 2½ to 5½ years) performed in Rochester. Conditioning techniques recommended by Myklebust, with frequencies 512, 1,024, 2,048 and 4,096 cps, with the use of pictures of a dog, cat, bird, and mouse, were employed, each child being tested individually; teams of two made it possible to speed up the examination. Each child who failed the screening test was given a more complete evaluation at the Rochester Hearing and Speech Society, consisting of a second screening test followed by a pure-tone threshold test and an otologic examination for children who failed the second screening test; in addition, all children who reported for the clinical evaluation were given a free-field speech-reception test. The percentage of children who could not be screened for one reason or another varied from 12.3% at age 2½-3½ to 3.3% at age 4½-5½ years. The average time required to condition and screen a child five and one-half minutes. Of the 431 children successfully screened, 6.5% were considered to have a hearing loss. Full details are given of the methods used for testing.

Finzi [58] contributes a paper on the results of audiometric pure-tone and speech tests in presbyacusis. The material used comprised 121 men and women between the ages of 60 and 93, divided for his purpose into three groups according to age. Tests included the usual air- and bone-conduction curves made on a Maico F-1 audiometer and discrimination tests made from a selected list of words recorded on phonograph disks. Recruitment tests by the Fowler method were also made in those cases where this was considered possible. From the results of his tests, which were indicative of a perceptive-hearing defect, he concluded, in agreement with Carhart and other authors, that presbyacusis is due to changes both in the peripheral and the central auditory organs. He regards these changes as a physiological degenerative process; in contradistinction to the lesions in younger persons, which are due to disease or toxic causes.

A study of hearing acuity in adult aphasic and cerebral-palsied subjects was made by Terr, Goetzinger, and Rousey.[59] Differences in hearing acuity for pure tones by air conduction between the normal-hearing group and the aphasic subjects were significant at the 1% level of confidence for all frequencies and for both the left and the right ears. This was also true for the cerebral-palsied subjects for frequencies 125, 500, and 1,000 cps. Differences for the higher frequencies were nonsignificant. This substantiates Salzman's observations that cerebral-palsied subjects of the spastic type tend to have low-frequency losses. The aphasias were the only group to show the differences at 1% level of confidence for both ears at frequencies 4,000 and 8,000 cps. There was no significant difference between the left and the right ears of any of the groups, even in those with cerebrovascular etiologies or whether the lesion was in the left-dominant or right-nondominant cerebral hemisphere. The hearing loss for both groups was similar, averaging less than 15 db. through the speech frequencies. The explanation for this may be the decreased ability of these persons to attend and concentrate, a possible concomitant of the particular types of brain injury studied, and decreased ability to differentiate the foreground signal in background-ambient noise.

In athetoids, the perceptive type of hearing is assumed to be due to retrocochlear damage. If this is so, then such patients might be expected to respond in an atypical way to a battery of audiologic tests which are sensitive to the detection of perceptive type of hearing loss with cochlear involvement. Ten athetoids with normal otological findings were tested with a battery of audiologic tests by Flottorp et al.[60] Each patient showed perceptive type of hearing loss more noticeable in the high frequencies, as well as signs of cochlear involvement; all were found to have loudness recruitment. On the basis of the outcome of the testing,

it was concluded that the hearing loss is of cochlear origin. This is contrary to the majority of opinions expressed by others. However, previous studies have not included as extensive a battery of audiologic tests. It is the writers' opinion that the signs of cochlear involvement in these athetoids may be explained on the possibility of oxygen deprivation as the causative factor.

O'Neill and Grimm[61] made a random-sampling hearing survey at a County Fair in Ohio during the summer of 1955. Every 10th person entering the fairgrounds was given a brochure stating the purpose, the nature and length of the test, and the age limitation—14 to 65 years. It had a pass enabling them to take the test. Only 224 of 1,226 receiving the brochures appeared for the test. Twenty-nine per cent, or 65, exhibited an average loss of hearing greater than 15 db. through the speech range for one or both ears. This far exceeds the 1% to 3% incidence of hearing loss found in the adult population. There were many more males than females, 138 to 86. The type of sampling technique employed in this study as well, as the results obtained in the Milwaukee Fair survey (Glorig et al.), is not too workable in terms of the obtaining of random representation of the hearing acuity of a selected population. The ideal type of sample should not be based solely on such concepts of population distribution but should also take into consideration other vital statistics of a particular locality, such as distribution by sex, occupation, and socioeconomic position.

Glorig[62] made a determination of the normal threshold of hearing by ear-phone listening on two groups of persons between the ages of 18 and 24 years. This was done to study the relations of normal hearing to present and proposed standard reference levels for normal hearing and to provide additional data which might explain differences in presently reported studies. His results agree very well with previous determinations when the conditions of the studies are comparable. He shows where the auditory-threshold data may vary, depending on such variables as education, practice effect, representatives chosen for the test, and ear pathology. The main differences occur when survey reports are compared to laboratory studies. When these two are compared, there is a difference in the threshold data with consistently lower values in the laboratory studies. When like studies are compared, the agreement is good, except for minor variations, which may be accounted for on the basis of technique and sampling. The question now to be resolved is whether survey or laboratory data should be used for establishing a normal-hearing reference level.

Hearing Aids

A clinical evaluation of monaural and binaural hearing aids was made by Markle and Aber.[63] They found the discrimination scores with binaural hearing aids were not significantly different from those obtained with monaural hearing aids when the test words were presented at a level of 10 db. above the intensity of noise. However, significantly better scores were obtained with binaural hearing aids when the test words were presented at the same intensity as the noise and at a level of 10 db. below the intensity of the noise. They conclude that when a subject is fitted with true binaural hearing aids he experiences significantly better discrimination than when exposed to a listening environment which is strongly influenced by a competitive background noise.

Hedgecock and Sheets[64] tested 30 hard of hearing persons for speech comprehension with monaural and binaural hearing aids of the eyeglass type. Differential results were not statistically significant. There appeared to be a trend favoring the binaural over the monaural aid in respect to speech comprehension in quiet surroundings. The study did not attempt to investigate the ability to localize sounds or the effect of noisy environments on performances of the binaural aids. The binaural fitting of a

hearing aid should be an individual matter. The demonstrated benefits should be reconciled against such other considerations as initial cost, differences in upkeep, and the relative discomfort of wearing two hearing aids.

Siegenthaler [65] reviewed the literature on the incidence of hearing-aid use in school children and the changes produced in speech-reception thresholds. He also gives data on hearing-aid use in a predominantly rural school district in Pennsylvania. About 1 elementary-grade child in 1,100 needs acoustic amplification, and about 1 second-grade child in 500 needs acoustic amplification. In the total school population of this district in Grades 1 to 12, about 1 child in 750 needs acoustic amplification. In practice, however, 1 child in 1,250 is using acoustic amplification. This difference is due to certain difficulties in providing every child who needs acoustic help with that kind of assistance. The average amount of acoustic gain for speech-reception threshold obtained by school children wearing hearing aids has been found by different investigators to be from 15 to over 40 db. Among this group the average gain was 22 db. This average gain results in about a 60% reduction in hearing loss as a result of wearing hearing aids.

Miscellaneous

The Subcommittee on Listing of Audiometers [66] sets down the principles and rules of procedure for this listing, which has been approved by the Committee on Conservation of Hearing. The objective of the procedure is to determine that a given model of audiometer is so designed and constructed that it can be calibrated by its manufacturers to conform to its appropriate standard. Several laboratories have been designated to carry out these testing procedures. The Committee will publish periodically a list of tested audiometers which meet with appropriate standards. It is implicit that the manufacturers will recognize the benefits to themselves of such a listing and will pay the fee necessary to secure the services of the testing laboratories. The plan for listing is presented in detail in the article.

Lehmann,[67] an engineer and member of the French National Centre for Telecommunication Study, contributes an article on the necessity for better standardization of hearing aids and audiometers in France. The criteria for gauging hearing-aid performance, which have been employed for a number of years in this country, are stated. Methods for standardization of audiometers as well as standards for normal thresholds by both air- and bone-conduction in different countries together with apparatus employed are described.

Davis and Usher [68] contribute a comprehensive discussion on the question of what should constitute our 0, or normal, audiometric hearing level. The present American Standard for this purpose is set forth in the American Standard for Audiometers for General Diagnostic Purposes, contained in a report published by the National Bureau of Standards, in 1951, and is accepted by the American Medical Association. It is based upon the hearing data obtained in the U. S. Public Health Survey of 1935-1936. Before that, a different 0 level had been established, about 30 years ago, for the Western Electric 2A Audiometer. The adequacy and accuracy of the present American standard has been questioned repeatedly, especially since the publication of recent British data that diverge significantly from those which served as a basis in our own country. The authors discuss the British, the Wisconsin, and other surveys, as well as the technical and other difficulties which impede the formation of an international 0 reference audiometric pure-tone standard. This is a thorough discussion of the subject which must be read in the original for those who are interested.

Reger and Kos [69] suggest a classification of ear or hearing involvements based solely on the anatomic localization of pathology. This is preferable from a diagnostic and treatment point of view. The classification

consists of three main categories: (1) tympanic, (2) cochlear, and (3) neural. Where cochlear and neural cannot be accurately differentiated, the term mediotympanic can be used until accurate localization is determined. This nomenclature is presented because it is based on a unified concept, namely, anatomic localization of pathology responsible for hearing involvements; it employs familiar terminology in a logical manner; it includes additional anatomic categories as more refined localization-testing techniques become available, and it encourages a more critical and exact determination of the localization of pathology which interferes with the hearing process.

Bioelectric examinations of the acoustic pupillary reflex in 10 rabbits were performed by Yamashita and Harano.[70] Microelectrodes were sunk into the sphincter and dilator muscles of the pupil (iris). Acoustic stimuli were then applied in dark rooms as well as in bright rooms and the status of the pupil observed. The acoustic reflex of the pupil was noticed to consist of two phases: a quick miosis and a slow mydriasis.

Certain facts could be deduced from these observations: Sudden acoustic stimuli from 1,000 to 2,000 cps, 100 phon, were found to be adequate stimuli for the acoustic pupillary reflex (sphincter as well as dilator reflex) in dark as well as in bright rooms; that in the dark room, generally, both iris muscles contracted reflectively, and that the latent periods for the sphincter reflex were shorter than for the dilator reflex; with adequate acoustic stimulation in a bright room (above 300 lux), the dilator reflex only appears because the pupils in a bright room are already miotic. The adaptation of the acoustic sphincter reflex in a dark room becomes clearly recognizable at a second stimulation with 1,000 cps, 100 phon. The sound-intensity-threshold values of the acoustic pupillary reflex in a partially illuminated room (at 30 lux) are different in the young rabbits from those in the old rabbits.

Mitrinowicz-Modrzejewska[71] performed electric testing of the vestibular apparatus on 50 subjects with the help of chronaxiametry. Small electrodes were placed on the eyelid or tragus of the same side, respectively. The vestibular apparatus was then tested with electric stimuli and the falling tendency observed.

The chronaxia values elicited in this manner from the eyelids were found to be essentially lower than those elicited from the tragus.

The conclusions drawn from these experiments by the author were that the electric stimuli applied at the tragus stimulated the peripheral vestibulum and also the peripheral labyrinth, while those applied at the eyelid stimulated the retrolabyrinthine nerve stem of the vestibular nerve. The chronaxia values attained at the tragus or eyelid, respectively, convey information regarding the status of the peripheral labyrinth or of the vestibular nerve stem.

Among other things, these findings may find application in the examination of deaf-mute children. They may be valuable in making a differential diagnosis between a true deaf-mutism and aphasia with hard of hearing, or a functional deaf-mutism, respectively.

111 N. Wabash Ave. (2).

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News and Comment

GENERAL NEWS

Awards Given at A. M. A. Meeting.—An exhibit entitled "Ear Surgery in 3-D: Tympanoplasty, Stapes and Fenestration," by Drs. J. Brown Farrior and Robert L. Levine, of Tampa, Fla., received the Billings gold medal award for excellence of correlating facts and presentation at the Annual Meeting of the American Medical Association, at Atlantic City, N. J.

The exhibit consists of a series of color slides to demonstrate the correct operation for a specific pathology as determined by surgery.

The exhibit was rebuilt after it was partially destroyed by a fire while being shown at the annual session of the American Academy of Opthalmology and Otolaryngology at the Palmer House in Chicago in October, 1958.

The Award of Merit of the American Medical Association Section on Laryngology, Otology, and Rhinology was presented to Drs. Joel Jay Pressman, Mildred Burtz Simon, and Walter Berman, of the City of Hope National Medical Center, Duarte, Calif., and the University of California at Los Angeles for their exhibit entitled "Primary Repair of Defects Following Removal of Tumors of the Face."

Honorable Mention was received by Drs. Chevalier L. Jackson, John V. Blady, Charles M. Norris, Robert Robbins, and Ernest L. McKenna, of Temple University Medical Center, Philadelphia, for their exhibit entitled "Cancer of the Larynx and Hypopharynx: Application of Clinical Classification and Staging in the Study of a Series of Cases."

Awards for Scientific Films.—Two international awards for excellence of a scientific film have been presented to Hans Von Leden, M.D., assistant professor of otolaryngology, Northwestern University Medical School, and Paul Moore, Ph.D., lecturer in the same department. The U. S. Department of State sent notice of the awards this week although the film festivals were held several months ago.

The film, "The Larynx and Voice—Laryngeal Physiology Under Daily Stress," was one of 320 entries in the Fourth International Review of Scientific Films, held in Rome. All branches of science were represented at the film festival.

Dr. Von Leden's film was awarded the Gold Medal of the Italian Red Cross and was one of the few films of a medical subject to receive a prize. This prize was presented to the United States representative in Rome and was the only major prize won by the United States at this festival.

The second award was won at the Third International Review for Scientific Education, at the University of Padua, under the sponsorship of the international film festival in Venice. Scientific films were judged in Padua. The award to the Northwestern scientists was for "education in the field of medicine." They were also presented with a bronze ox head—the traditional gift at the University of Padua for awards of merit.

During the past three years, Von Leden and Moore have won five international awards for their work in laryngeal physiology. The motion pictures, made in the laryngeal Research Laboratory of the William and Harriet Gould foundation, provide a new method of studying the function of the larynx by ultra high-speed photography and give a graphic demonstration of changes in the larynx during periods of normal activity and during stress and strain. This method aids in early diagnosis and treatment of larnygeal disorders. The films have also disclosed some previously unknown facts on laryngeal physiology.

The camera equipment and the system of mirrors by which the image is reflected from the throat to the camera were designed under the direction of Dr. Moore.

Call for New and Used Medical Textbooks.—The Polish Institute of Arts and Sciences in America has issued a call to all American medical groups, colleges, hospitals, and physicians to contribute new and used medical textbooks, reference books, and journals to medical schools and teaching hospitals. In the plea for contributions of medical books and reference books, the chairman of the project, Mr. Maryan Chodacki, said, "The

effects of the devastations of war upon medical education in Poland are far from overcome even though the fighting in Poland ended 15 years ago. There are many physicians and medical students in Poland who read English and who are avid for American medical books and journals. Following the long traditions of American Medicine which has assisted so many other war devasted nations in rebuilding their medical education programs, American physicians, medical groups, colleges, and hospitals can now send their contributions through the Polish Institute of Arts and Sciences in America. This organization is prepared to act as *a receiving center for such contributions and to pay all shipping costs."*

Individual persons and organizations may send books and journals prepaid postage, express or freight, to Mr. Maryan Chodacki, Polish Institute of Arts and Sciences in America, 145 E. 53d St., New York 22. Persons and groups with contributions but without funds to pay shipping costs to New York should write to Mr. Chodacki.

ANNOUNCEMENTS

Meeting of American Association for Cleft Palate Rehabilitation.—The American Association of Cleft Palate Rehabilitation will be holding its 18th Annual Convention at the Brown Palace Hotel, in Denver, on Thursday, Friday, and Saturday, May 12, 13, and 14, 1960.

This Association is composed of medical, dental, and paramedical specialists who are interested in the rehabilitation of persons with cleft lip and palates.

Books

BOOKS RECEIVED

Cliniques Radiologiques (Première série: L'Agrandissment radiographique direct dans l'examen de la base du crane et des micro-fractures du rocher. By Prof. J. Roussel, with collaboration of P. Schoumacher, M. Pernot, and R. Poirè. Price, 3.200 fr. Pp. 102, with 59 illustrations. Masson & Cie, 120 boulevard Saint-Germain, Paris 6°, 1959.

Ciba Foundation Symposium: Regulation of Cell Metabolism. Edited by G. E. W. Wolstenholme, O.B.E., M.A., B.Ch., and Cecilia M. O'Connor, B.Sc. Price, $9.50. Pp. 387, with many illustrations, charts, and tables. Little, Brown & Company, 34 Boston St., Boston, 1959.

Ciba Foundation Symposium: Carcinogenesis: Mechanisms of Action. Edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Maeve O'Connor, B.A. Price, $9.50. Pp. 336, with many illustrations, charts, and tables. Little, Brown & Company, 34 Beacon St., Boston, 1959.

BOOK REVIEWS

Surgery of the Ear. By George E. Shambaugh, M.D. Price, $27.50. Pp. 669, with 326 illustrations. W. B. Saunders Company, 218 W. Washington Sq., Philadelphia 5, 1959.

This book by an eminent otologist is of outstanding scope and importance. Its scientific reliability and complete coverage from basic fundamentals of otologic surgery to the niceties of newest techniques undoubtly make it a classic. The illustrations are informative, simple, and very clear. The conciseness and simplicity of style indicate meticulous editing, mature consideration, and vast experience.

The twenty-two chapters cover practically all information of importance to otologic surgery and are informative both to residents and to accomplished surgeons. Each chapter is based on the newest concepts and is almost extraordinary in its own right. The first few chapters cover the development of the ear, its anatomy (particularly well presented), and an especially fine presentation on radiologic examination. The next section contains excellent and detailed description on all phases of surgery for ear infection. Modern concepts and techniques of mastoid surgery are definitively presented. The third, and most timely, section deals with surgery for deafness. It is a brillantly composed series starting with the mechanism of hearing and covering congenital defects, typmpanoplastic procedures, stapes mobilization, and fenestration. The new techniques of mobilization and footplate fenestration are described and cautiously evaluated. The diagrams in this section are especially commendable for their simplicity and effectiveness.

The last section concerns itself with surgery of the facial nerve, tumors, and Ménière's disease. Descriptions of the indications, techniques, and results are interestingly and reliably presented.

Throughout the book are the series of carefully selected annotated pictures of historically important persons in the field of otologic surgery. This adds a very fine tenure to the book and is definitely an asset.

There is little doubt that this book is one of the most important major contributions in the field of otolaryngology.

Die Speichel drüsen des Menschen (The Salivary Glands in Humans.) By S. Rauch. Price, $18.80. Pp. 506, with 227 illustrations. Georg Thieme Verlag, Herdweg 63, (14a) Stuttgart, Germany; Intercontonental Medical Book Corporation, 381 4th Ave., New York 16, 1959.

This excellent book deals with every problem that may involve the salivary glands. The anatomy, histology, and embryology are first discussed in great detail. Then, in the chapter on physiology, the author presents the various methods of examination and the chemical analysis under both normal conditions and various forms of stimulation. The function of the glands and their relationship to the general body functions and to metabolism are amply discussed.

In the chapter on pathology, the author presents three general conditions. 1. Inflammatory diseases of the glands; these are subdivided into the bacterial, mycotic, viral, or aseptic categories. 2. Alterations in the structure of the glands; these include allergy, hormonal influences, neurogenic influences, and other indigenous structural changes of various etiologies. 3. The various new growths—both benign and malignant—are presented in great detail.

In addition, there are chapters dealing with calculi and anomalies, and finally there is a detailed discussion of both medical and surgical treatment of the pathologic conditions.

There is an extensive bibliography after each chapter. This book is strongly recommended for the general practitioner, the general surgeon, the oral surgeon, and the otolaryngologist.

Il cortisone e le sostanze ad azione cortisonica in otorinolaringoiatria: Ricerche sperimentali e cliniche. By Oscar Sala. Price, not given. Pp. 367, with 138 illustrations. Minerva Otorinolaringologica, Padova, Italy, 1959.

After a general survey on structure and physiology of the adrenal glands and their relation to the hypophysis, the influence of the cortisones on some tissue groups—connective, vascular, lymphatic—is discussed. After the basic exposition, the three main chapters report on the role of the steroids in aural, nasal, and laryngeal pathology, respectively. Nasopharyngeal fibroma and caustic lesion of the esophagus are treated in special sections. Many illustrations (some colored) and several charts accompany the text, which is followed by a forty-page list of references.

The presentation is built on a wide field of personal experience—experimental and clinical. The extensive experimental work of the author, after having been previously reported in all detail, is summarized to serve as a backbone to the analysis. Clinical procedure is given in a precise manner which will be easy to follow, e. g., in the pre- and postoperative care in microsurgery of the ear.

Since it comes from the department of M. Arslan, in Padova, long a center of investigations in collagenous diseases, this monograph can be considered the authoritative one on the much-debated problems regarding the role of the steroids in otolaryngology. In each chapter, close attention is given to surveys of the literature. In addition to departments of otolaryngology and endocrinology, where the book will be indispensable, workers in many other units will find hard-to-come-by information by its perusal.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. G. Arnold Henry, 170 St. George St., Toronto.
Secretary: Dr. Donald M. MacRae, 324 Spring Garden Rd., Halifax, N. S.
Meeting: Sheraton-Brock Hotel, Niagara Falls, Ontario, Oct. 9-10, 1959.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Association of Allergology

President: Dr. Samuel M. Feinberg, 303 E. Chicago Ave., Chicago 11.
Secretary-General: Dr. José Quintero Fossas, Paseo 313, Vedado, Havana, Cuba

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. John R. Lindsay, 950 E. 58th St., Chicago.
Secretary-Treasurer: Dr. Louis Savitt, 3403 W. Lawrence Ave., Chicago 25.
Meeting: 24th Annual Congress, Palmer House, Chicago, Sept. 14-18, 1959.

International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

Mexican Society of Otolaryngology

President: Dr. Rafael Giorgana
Secretary: Dr. Carlos Valenzuela, Monterrey 47-201, Mexico 7, D. F.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Paul H. Holinger, Chicago.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Victor R. Alfaro, Washington, D. C.
Secretary: Dr. Walter E. Heck, San Francisco.

American Academy of Ophthalmology and Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

American Broncho-Esophagological Association

President: Dr. Verling K. Hart, 106 W. 7th St., Charlotte, N. C.
Secretary: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3.
Meeting: Deauville Hotel, Miami Beach, Fla., March 15-16, 1960.

American Laryngological Association

President: Dr. William J. McNally, Montreal.
Secretary: Dr. Lyman G. Richards, Wellesley Hills, Mass.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Meeting: Deauville Hotel, Miami Beach, Fla., March 18-19, 1960.

SECTIONS:

Eastern.—Chairman: Dr. Joseph L. Goldman, New York.
Southern.—Chairman: Dr. James T. King, Atlanta, Ga.
Middle.—Chairman: Dr. Alfred J. Cone, St. Louis, Mo.
Western.—Chairman: Dr. Robert C. McNaught, San Francisco, Calif.

American Rhinologic Society

President: Dr. Kenneth H. Hinderer, 402 Medical Arts Building, Pittsburgh 13.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.
Annual Clinical Session: Oct. 8-9, 1959, Illinois Masonic Hospital, Chicago.
Annual Meeting: Oct. 10, 1959, Belmont Hotel, Chicago.

American Otological Society, Inc.

President: Dr. Robert C. Martin, 384 Post St., San Francisco 8.
Vice-President: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.
Meeting: March 13-14, 1960.

American Otorhinologic Society for Plastic Surgery, Inc.

President: Dr. Raymond S. Rosedale, 610 N. Market Ave., Canton 2, Ohio.
Secretary-Treasurer: Dr. Joseph G. Gilbert, 75 Barberry Lane, Roslyn Heights, N. Y.
Meeting: Oct. 11, 1959, Conrad Hilton Hotel, Chicago, and March 6-13, 1960, Deauville Hotel, Miami Beach, Fla.

American Society of Facial Plastic Surgery

President: Dr. Oscar J. Becker, Chicago.
Vice-President: Dr. Sam H. Sanders, Memphis.
Secretary: Dr. Samuel M. Bloom, 123 E. 83d St., New York 28.
Treasurer: Dr. Joseph C. Miceli, Brooklyn.
Meeting: Oct. 15, 1959, Chicago.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N.Y.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6.

Otosclerosis Study Group

President: Dr. E. P. Fowler Jr., 180 Fort Washington Ave., New York 32.
Secretary-Treasurer: Dr. Arthur L. Juers, 1018 Brown Building, Louisville 2.

Society of Military Otolaryngologists

President: Lt. Col. Stanley H. Bear (MC), USAF, Maxwell AFB, Ala.
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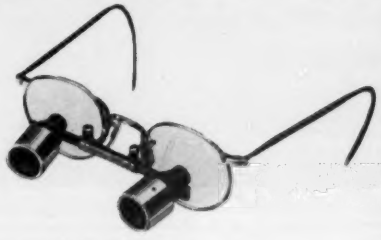

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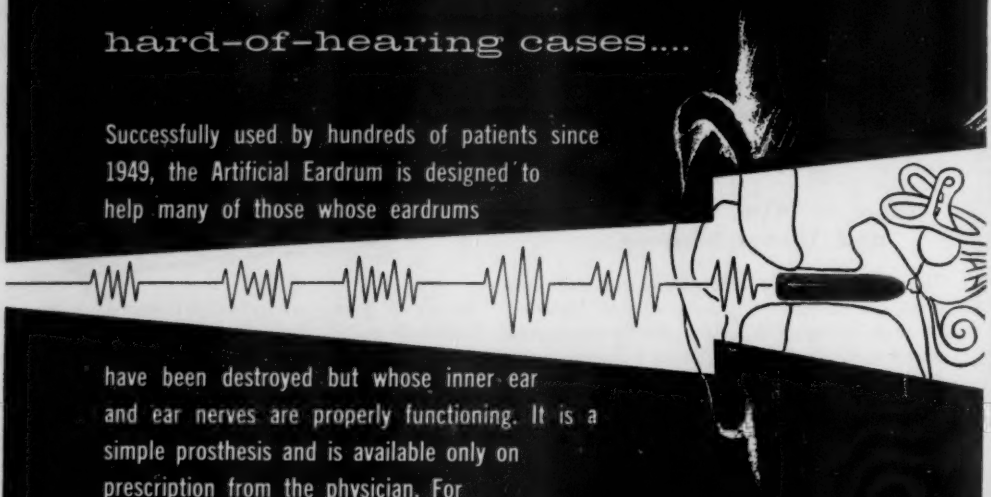

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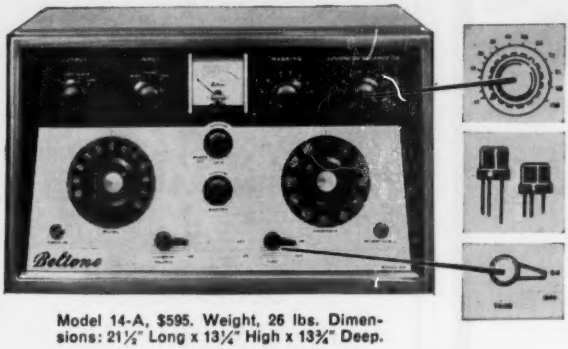

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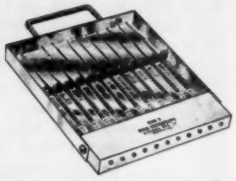

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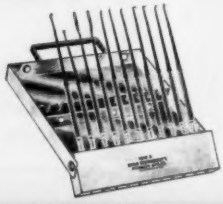

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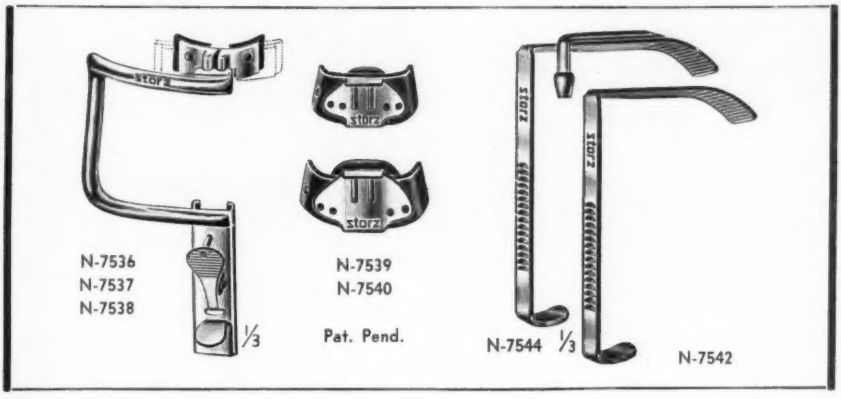

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